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☐	L6	l1 and L5	99
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☐	L4	L1 same (plant or fung$ or yeast or algae or insect)	24
☐	L3	L1 and (plant or fung$ or yeast or algae or insect)	188
☐	L2	L1 and lower eukary$	33
☐	L1	N-acetylglucosaminyltransferase III or GnTIII or GnT III	232

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L2        682 L1 (3A) (II OR III)

=> s l2 and cell?
L3        485 L2 AND CELL?

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L5  ANSWER 1 OF 206  EMBASE  COPYRIGHT (c) 2007 Elsevier B.V. All rights
    reserved on STN
AN  2005504944  EMBASE  <<LOGINID::20070409>>
TI  Reduction of the Gal-.alpha.1,3-Gal epitope of mouse endothelial
      ***cells***  by transfection with the N-  ***acetylglucosaminyltransferas***
  ***    e***    ***III***  gene.
AU  Chung T.-W.; Kim K.-S.; Kim C.-H.
CS  C.-H. Kim, National Research Laboratory for Glycobiology, Department of
    Biochemistry and Molecular Biology, Dongguk University College of Oriental
    Medicine, Kyungju 780-714, Korea, Republic of. chkimbio@dongguk.ac.kr
SO  Molecules and Cells, (2003) Vol. 16, No. 3, pp. 368-376. .
    Refs: 51
    ISSN: 1016-8478  CODEN: MOCEEK
CY  Germany
DT  Journal; Article
FS  022  Human Genetics
    029  Clinical Biochemistry
LA  English
SL  English
ED  Entered STN: 22 Dec 2005
    Last Updated on STN: 22 Dec 2005
AB  In order to prevent hyperacute rejection in pig-to-human
    xenotransplantation, it would be very useful to be able to down-regulate
    the Gal .alpha.1-3 Gal.beta. 1-4 GlcNAc-R (.alpha.-Gal epitope) in mouse
    and swine tissues. When the .beta.-D-mannoside .beta.-1,4-N-
    acetylglucosaminyl-transferase III (GnT-III) gene was introduced into
    mouse aorta endothelial  ***cells***  (MEC) their susceptibility to
    complement-mediated  ***cell***  lysis by normal human serum (NHS) was
    reduced. Expression of GnT-III also suppressed the antigenicity of MEC to
    human natural antibodies as shown by binding of Griffoniac simplicifolia 1
    isolectin (GS1B4 lectin) to the .alpha.-Gal epitope. Western blot
    analysis indicated that the reactivity of the glycoproteins of the
    transfectants to NHS and GSIB4 lectin was reduced to approximately the
    same extent. Thus GnT-III, a key enzyme involved in the formation of
    branched N-linked sugars, reduces the expression of xenoantigens,
    suggesting that this approach may be of value in clinical
    xenotransplantation. .COPYRGT.KSMCB 2003.

L5  ANSWER 2 OF 206  EMBASE  COPYRIGHT (c) 2007 Elsevier B.V. All rights
    reserved on STN
AN  2005504941  EMBASE  <<LOGINID::20070409>>
TI  Remodeling of the major mouse xenoantigen, Gal.alpha.1-3Gal.beta.1-
4GlcNAc-
      R, by N-  ***acetylglucosaminyltransferase***  -  ***III***  .
AU  Chung T.-W.; Kim K.-S.; Kang S.-K.; Lee J.-W.; Song E.-Y.; Chung T.-H.;
    Yeom Y.-I.; Kim C.-H.
CS  C.-H. Kim, National Research Laboratory for Glycobiology, Department of
    Biochemistry and Molecular Biology, Dongguk University COM, Kyungju
    780-714, Korea, Republic of. chkimbio@dongguk.ac.kr
SO  Molecules and Cells, (2003) Vol. 16, No. 3, pp. 343-353. .
    Refs: 60
    ISSN: 1016-8478  CODEN: MOCEEK
CY  Germany
DT  Journal; Article
FS  026  Immunology, Serology and Transplantation
    029  Clinical Biochemistry
LA  English
SL  English
ED  Entered STN: 22 Dec 2005
    Last Updated on STN: 22 Dec 2005
AB  .beta.-D-Mannoside .beta.-1,4-N-  ***acetylglucosaminyltransferase***
      ***III***  (GnT-  ***III***  ) catalyses the attachment of an
    N-acetylglucosamine (GlcNAc) residue to mannose in the .beta.(1-4)
    configuration in N-glycans, and forms a bisecting GlcNAc. We have
    generated transgenic mice that contain the human GnT-III gene under the
    control of the mouse albumin enhancer/promoter [Lee et al., (2003)].
    Overexpression of this gene in mice reduced the antigenicity of N-glycans
    to human natural antibodies, especially in the case of the .alpha.-Gal
    epitope, Gal.alpha.1-3Gal.beta.1-4GlcNAc-R. Study of endothelial
      ***cells***  from the GnT-III transgenic mice revealed a significant
    reduction in antigenicity, and a dramatic decrease in both complement- and
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natural killer ***cell*** -mediated mouse ***cell*** lysis. Changes in the enzymatic activities of other glycosyltransferases, such as .alpha.1,3-galactosyltransferase, and .alpha.-6-D-mannoside .beta.-1,6 N-acetylglucosaminyltransferase V, did not point to any interaction between GnT-III and these enzymes in the transgenic mice, suggesting that this approach may be useful in clinical xenotransplantation. .COPYRGT.KSMCB 2003.

L5 ANSWER 3 OF 206 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2004334373 EMBASE <<LOGINID::20070409>>
TI Role of N-glycans in growth factor signaling.
AU Takahashi M.; Tsuda T.; Ikeda Y.; Honke K.; Taniguchi N.
CS N. Taniguchi, Department of Biochemistry, Osaka Univerisity, Graduate School of Medicine, B1, 2-2 Yamadaoka, Suita, Osaka 565-0871, Japan. proftani@biochem.med.osaka-u.ac.jp
SO Glycoconjugate Journal, (2003) Vol. 20, No. 3, pp. 207-212. . Refs: 43 ISSN: 0282-0080 CODEN: GLJOEW
CY Netherlands
DT Journal; General Review
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 26 Aug 2004 Last Updated on STN: 26 Aug 2004
AB Secreted proteins and membrane proteins are frequently post-translationally modified by oligosaccharides. Therefore, many glycoproteins are involved in signal transduction. One example is growth factor receptors, which are membrane proteins that often contain oligosaccharides. The oligosaccharides in those growth factor receptors play crucial roles in receptor functions. An analysis of glycosyltransferase-transfectants revealed that the branching structures of oligosaccharide also serve as important determinants. For example, N-glycans of epidermal growth factor receptor (EGFR) are involved in receptor sorting, ligand binding and dimerization. The addition of a bisecting GlcNAc to N-glycans increases the endocytosis of EGFR. N-glycans of Trk, a high affinity nerve growth factor receptor, also affect its function. Thus, oligosaccharides play an important role in growth factor signaling.

L5 ANSWER 4 OF 206 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2003509322 EMBASE <<LOGINID::20070409>>
TI Determination of UDP-N-acetylglucosamine:.beta.-D-mannoside-1,4-N-***acetylglucosaminyltransferase*** - ***III*** in patients sera with chronic hepatitis and liver cirrhosis using a monoclonal antibody.
AU Song E.-Y.; Kim K.-S.; Kim K.-A.; Kim Y.-D.; Kwon D.-H.; Byun S.-M.; Kim H.-J.; Chung T.-W.; Choe Y.-K.; Chung T.-W.; Kim C.-H.
CS C.-H. Kim, Natl. Res. Lab. of Glycobiology, Dept. of Biochem. and Molec. Biology, Dongguk University COM, Sukjang-Dong 707, Kyungju, Kyungbuk 780-714, Korea, Republic of. chkimbio@dongguk.ac.kr
SO Glycoconjugate Journal, (2002) Vol. 19, No. 6, pp. 415-421. . Refs: 24 ISSN: 0282-0080 CODEN: GLJOEW
CY Netherlands
DT Journal; Article
FS 029 Clinical Biochemistry
048 Gastroenterology
LA English
SL English
ED Entered STN: 30 Dec 2003 Last Updated on STN: 30 Dec 2003
AB The glycoprotein UDP-N-acetylglucosamine:.beta.-D-mannoside-1,4-N-***acetylglucosaminyltransferase*** - ***III*** (GnT- ***III***) catalyzes the addition of N-acetylglucosamine via a .beta.-1,4-linkage to the .beta.-linked mannose of the trimannosyl core of N-linked glycans. It has been reported that the expression of GnT-III increases in many oncogenically transformed ***cells*** and human hepatocellular carcinoma (HCC) tissues, and GnT-III enzyme activity in serum can be used for the detection and monitoring of primary hepatomas and hepatocellular carcinomas. A solid-phase enzyme-linked immunosorbent sandwich assay in which a polyclonal antibody (PAb) to aglycosylrecombinant GnT-III (AGR-GnT-III) and a monoclonal antibody (mAb) are employed as a capture protein and probe protein, respectively, is described. The sensitivity of the PAb-mAb sandwich assay, as determined by the dose-response effect for AGR-GnT-III, was 10 ng/ml. This assay was specific for GnT-III and did not detect .beta.-1, 6-N-acetylglucosaminyltrasferase-V (GnT-V). AGR-GnT-III concentrations in 377 serum specimens were determined by the PAb-mAb sandwich assay and the results were analyzed based on the disease category, using 1.99 .mu.g/mL (AGR-GnT-III) as a cut-off value. The AGR-GnT-III level of 61 normal serum samples was 0.57 .+-. 0.71 .mu.g/ml (mean .+-. SD). The results revealed an elevation in serum AGR-GnT-III levels in 60 of 86 patients (3.03 .+-. 2.04 .mu.g/ml) with liver cirrhosis (LC) and 86 of 91 patients (2.73 .+-. 0.59 .mu.g/ml) with chronic hepatitis (CH). By contrast, 3 of 61 normal subjects, 9 of 34 patients (1.02 .+-. 1.03 .mu.g/ml) with acute hepatitis and 8 of 38 patients (1.79 .+-. 0.56 .mu.g/ml) with a variety of non-hepatic diseases exhibited a slight increase above the cut-off value. These results indicate that serum AGR-GnT-III levels are elevated predominantly in LC or CH cases. Serum AGR-GnT-III concentration, as measured by the developed PAb-mAb sandwich assay, may be a useful differential marker as a diagnostic aid for CH and/or LC and warrants further investigations with expanded serum panels.

L5 ANSWER 5 OF 206 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2003487756 EMBASE <<LOGINID::20070409>>
TI Reduced Hepatocyte Proliferation is the Basis of Retarded Liver Tumor Progression and Liver Regeneration in Mice Lacking N-***Acetylglucosaminyltransferase*** ***III*** .
AU Yang X.; Tang J.; Rogler C.E.; Stanley P.
CS P. Stanley, Department of Cell Biology, Albert Einstein College of Medicine, 1300 Morris Park Avenue, New York, NY 10461, United States. stanley@aecom.yu.edu
SO Cancer Research, (15 Nov 2003) Vol. 63, No. 22, pp. 7753-7759. . Refs: 33 ISSN: 0008-5472 CODEN: CNREA8
CY United States
DT Journal; Article
FS 016 Cancer
029 Clinical Biochemistry
048 Gastroenterology
LA English
SL English
ED Entered STN: 5 Jan 2004 Last Updated on STN: 5 Jan 2004
AB Mice lacking N- ***acetylglucosaminyltransferase*** ***III*** (GlcNAc-TIII) exhibit slightly but significantly retarded liver tumor progression after a single injection of 10 .mu.g/g diethylnitrosamine (DEN) and continued administration of phenobarbital (PB) in drinking water. A key question is whether the absence of GlcNAc-TIII inhibits ***cell*** proliferation or induces apoptosis. Because PB aids tumor progression, we tested whether it diminished the difference in tumor progression between Mgat3(+/+) and Mgat3(.DELTA./.DELTA.) mice. Here, we show that in the absence of PB, control males developed about twice as many liver tumor nodules as males lacking GlcNAc-TIII. Both the size of liver tumors and liver weights were significantly greater in DEN-treated wild-type or heterozygous mice. Apoptosis assays performed monthly after DEN treatment showed no differences between mutant and wild-type. However, there was a marked retardation in liver regeneration after partial (70%) hepatectomy (PH). Wild-type mice incorporated bromodeoxyuridine in .apprx.15% of hepatocyte nuclei at 48 h after PH, whereas mice lacking GlcNAc-TIII had only .apprx.5% positive nuclei. This was not because of enhanced apoptosis in mutant mice after PH. Expression of the Mgat3 gene remained undetectable in wild-type liver by Northern analysis after tumor induction or after PH. In addition, transgenic overexpression of GlcNAc-TIII in hepatocytes did not enhance tumor progression in Mgat3(.DELTA./.DELTA.) mice, and there were no differences in tumor progression or liver regeneration after PH between control and transgenic mice overexpressing GlcNAc-TIII in liver. Therefore, the nonhepatic action of GlcNAc-TIII promotes hepatocyte proliferation after PH, as well as the progression of DEN-induced tumors, providing evidence for a functional role of the bisecting GlcNAc on circulating glycoprotein growth factor(s) that stimulate hepatocyte proliferation.

L5 ANSWER 6 OF 206 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2003458216 EMBASE <<LOGINID::20070409>>
TI Overexpression of N- ***Acetylglucosaminyltransferase*** ***III*** Enhances the Epidermal Growth Factor-induced Phosphorylation of ERK in HeLaS3 ***Cells*** by Up-regulation of the Internalization Rate of the Receptors.
AU Sato Y.; Takahashi M.; Shibukawa Y.; Jain S.K.; Hamaoka R.; Miyagawa J.-I.; Yaginuma Y.; Honke K.; Ishikawa M.; Taniguchi N.
CS N. Taniguchi, Department of Biochemistry, Osaka Univ. Grad. School of Medicine, 2-2 Yamadaoka, Suita, Osaka 565-0871, Japan. proftani@biochem.med.osaka-u.ac.jp
SO Journal of Biological Chemistry, (13 Apr 2001) Vol. 276, No. 15, pp. 11956-11962. . Refs: 48 ISSN: 0021-9258 CODEN: JBCHA3
CY United States
DT Journal; Article
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 4 Dec 2003 Last Updated on STN: 4 Dec 2003
AB N- ***Acetylglucosaminyltransferase*** ***III*** (GnT- ***III***) is a key enzyme that inhibits the extension of N-glycans by introducing a bisecting N-acetylglucosamine residue. In this study we investigated the effect of GnT-III on epidermal growth factor (EGF) signaling in HeLaS3 ***cells*** . Although the binding of EGF to the epidermal growth factor receptor (EGFR) was decreased in GnT-III transfectants to a level of about 60% of control ***cells*** , the EGF-induced activation of extracellular signal-regulated kinase (ERK) in GnT-III transfectants was enhanced to .apprx.1.4-fold that of the control ***cells*** . A binding analysis revealed that only low affinity binding of EGF was decreased in the GnT-III transfectants, whereas high affinity binding, which is considered to be responsible for the downstream signaling, was not altered. EGF-induced autophosphorylation and dimerization of the EGFR in the GnT-III transfectants were the same levels as found in the controls. The internalization rate of EGFR was, however, enhanced in the GnT-III transfectants as judged by the uptake of (125)I-EGF and Oregon Green-labeled EGF. When the EGFR internalization was delayed by

dansylcadaverine, the up-regulation of ERK phosphorylation in GnT-III transfectants was completely suppressed to the same level as control ***cells*** . These results suggest that GnT-III overexpression in HeLaS3 ***cells*** resulted in an enhancement of EGF-induced ERK phosphorylation at least in part by the upregulation of the endocytosis of EGFR.

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AN 2003457597 EMBASE <<LOGINID::20070409>>
TI Down-regulation of the .alpha.-Gal epitope expression in N-Glycans of swine endothelial ***cells*** by transfection with the N-***Acetylglucosaminyltransferase*** ***III*** Gene: Modulation of the biosynthesis of terminal structures by a bisecting GlcNAc.
AU Koyota S.; Ikeda Y.; Miyagawa S.; Ihara H.; Koma M.; Honke K.; Shirakura R.; Taniguchi N.
CS N. Taniguchi, Department of Biochemistry, Osaka University Medical School, 2-2 Yamadaoka, Suita 565-0871, Osaka, Japan. proftani@biochem.med.osaka-u.ac.jp
SO Journal of Biological Chemistry, (31 Aug 2001) Vol. 276, No. 35, pp. 32867-32874. .
Refs: 56
ISSN: 0021-9258 CODEN: JBCHA3
CY United States
DT Journal; Article
FS 026 Immunology, Serology and Transplantation
029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 4 Dec 2003
Last Updated on STN: 4 Dec 2003
AB The down-regulation of the .alpha.-Gal epitope (Gal.alpha.1,3Gal.beta.3-R) in swine tissues would be highly desirable, in terms of preventing hyperacute rejection in pig-to-human xenotransplantation. In an earlier study, we reported that the introduction of the .beta.1,4-N-***acetylglucosaminyltransferase*** (GnT) ***III*** gene into swine endothelial ***cells*** resulted in a substantial reduction in the expression of the .alpha.-Gal epitope. In this study, we report on the mechanism for this down-regulation of the .alpha.-Gal epitope by means of structural and kinetic analyses. The structural analyses revealed that the amount of N-linked oligosaccharides bearing the .alpha.-Gal epitopes in the GnT-III-transfected ***cells*** was less than 10% that in parental ***cells*** , due to the alteration of the terminal structures as well as a decrease in branch formation. In addition, it appeared that the addition of a bisecting GlcNAc, which is catalyzed by GnT-III, leads to a more efficient sialylation rather than .alpha.-galactosylation. In vitro kinetic analyses showed that the bisecting GlcNAc has an inhibitory effect on .alpha.-galactosylation, but does not significantly affect the sialylation. These results suggest that the bisecting GlcNAc in the core is capable of modifying the biosynthesis of the terminal structures via its differential effects on the capping glycosyltransferase reactions. The findings may contribute to the development of a novel strategy to eliminate carbohydrate xenoantigens.

L5 ANSWER 8 OF 206 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2003452200 EMBASE <<LOGINID::20070409>>
TI Remodeling of the Major Pig Xenoantigen by N-***Acetylglucosaminyltransferase*** ***III*** in Transgenic Pig.
AU Miyagawa S.; Murakami H.; Takahagi Y.; Nakai R.; Yamada M.; Murase A.; Koyota S.; Koma M.; Matsunami K.; Fukuta D.; Fujimura T.; Shigehisa T.; Okabe M.; Nagashima H.; Shirakura R.; Taniguchi N.
CS S. Miyagawa, Division of Organ Transplantation, Department of Regenerative Medicine, Osaka Univ. Grad. School of Medicine, 2-2 Yamadaoka, Suita, Osaka 565-0871, Japan. miyagawa@orgtrp.med.osaka-u.ac.jp
SO Journal of Biological Chemistry, (19 Oct 2001) Vol. 276, No. 42, pp. 39310-39319. .
Refs: 56
ISSN: 0021-9258 CODEN: JBCHA3
CY United States
DT Journal; Article
FS 026 Immunology, Serology and Transplantation
029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 11 Dec 2003
Last Updated on STN: 11 Dec 2003
AB We have been successful in generating several lines of transgenic mice and pigs that contain the human .beta.-D-mannoside .beta. -1,4-N-***acetylglucosaminyltransferase*** ***III*** (GnT- ***III***) gene. The overexpression of the GnT-III gene in mice and pigs reduced their antigenicity to human natural antibodies, especially the Gal.alpha.1-3Gal.beta.1-4Glc-NAc-R, as evidenced by immunohistochemical analysis. Endothelial ***cell*** studies from the GnT-III transgenic pigs also revealed a significant down-regulation in antigenicity, including Hanganutziu-Deicher antigen, and dramatic reductions in both the complement- and natural killer ***cell*** -mediated pig ***cell*** lyses. Changes in the enzymatic activities of other glycosyltransferases, such as .alpha. 1,3-galactosyltransferase, GnT-IV, and GnT-V, did not support cross-talk between GnT-III and these enzymes in the transgenic animals. In addition, we demonstrated the effect of GnT-III in down-regulating the xenoantigen of pig heart grafts, using a pig to cynomolgus monkey transplantation model, suggesting that this approach may be useful in clinical xenotransplantation in the future.

L5 ANSWER 9 OF 206 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2003429715 EMBASE <<LOGINID::20070409>>
TI Sensitivity to human serum of gammaretroviruses produced from pig endothelial ***cells*** transduced with glycosyltransferase genes.
AU Kurihara T.; Miyazawa T.; Miyagawa S.; Tomonaga K.; Hazama K.; Yamada J.;
Shirakura R.; Matsuura Y.
CS Dr. T. Miyazawa, Res. Ctr. for Emerging Infect. Dis., Res. Inst. for Microbial Diseases, Osaka University, 3-1 Yamadaoka, Suita, Osaka 565-0871, Japan. takavet@biken.osaka-u.ac.jp
SO Xenotransplantation, (2003) Vol. 10, No. 6, pp. 562-568. .
Refs: 34
ISSN: 0908-665X CODEN: XENOFL
CY United Kingdom
DT Journal; Article
FS 004 Microbiology
026 Immunology, Serology and Transplantation
LA English
SL English
ED Entered STN: 6 Nov 2003
Last Updated on STN: 6 Nov 2003
AB Reduction of pig ***cell*** -surface .alpha.-galactosyl (Gal) epitope, Gal.alpha.1, 3Gal.beta.1, 4GlcNAc-R, by the introduction of glycosyltransferase genes is effective in suppressing hyperacute rejection (HAR) in pig-to-human xenotransplantation. The transmission of porcine endogenous retroviruses (PERVs) has been recognized as a potential risk factor associated with xenotransplantation. In this study, effects of the introduction of glycosyltransferase genes to pig ***cells*** on the sensitivity of gammaretroviruses to human serum were investigated. Pig endothelial ***cells*** (PEC), PEC transduced with .alpha.1,2 fucosyltransferase (FT), .alpha.2,3 sialyltransferase (ST) or N-***acetylglucosaminyltransferase*** ***III*** (GnT- ***III***), and human embryonic kidney (HEK) 293 ***cells*** were transduced with the LacZ gene with the packaging signal of murine leukemia virus (MuLV) under the control of the long terminal repeat of MuLV by a pseudotype infection. Then, the ***cells*** were further infected with PERV subtype B (PERV-B) or feline leukemia virus subgroup B (FeLV-B). Culture supernatants of the infected ***cells*** were mixed with human serum (HS) and then inoculated to HEK293 ***cells*** . The inoculated ***cells*** were histochemically stained and lacZ-positive blue foci were counted. Glycosyltransferase activity, xenoantigenicity, and .alpha.-Gal epitope density in the ***cells*** were measured at the time of the infection experiments. PERV-B or FeLV-B particles from the parental PEC were efficiently neutralized by HS, while those from PEC transduced with .alpha.1,2FT, .alpha.2,3ST or GnT-III were less sensitive to HS. The transduced PEC exhibited high levels of activity of the introduced glycosyltransferases, and expressed fewer xenoantigens and ***cell*** -surface .alpha.-Gal epitopes. Our results suggest that gammaretroviruses including PERVs produced by transgenic pigs, that are genetically modified to reduce the ***cell*** -surface .alpha.-Gal epitope to overcome the HAR in xenotransplantation, are less sensitive to HS.

L5 ANSWER 10 OF 206 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2003288082 EMBASE <<LOGINID::20070409>>
TI Antibodies that recognize bisected complex N-glycans on ***cell*** surface glycoproteins can be made in mice lacking N-***acetylglucosaminyltransferase*** ***III*** .
AU Lee J.; Park S.-H.; Stanley P.
CS Dr. P. Stanley, Department of Cell Biology, Albert Einstein College of Medicine, New York, NY 10461, United States. stanley@aecom.yu.edu
SO Glycoconjugate Journal, (1 Mar 2003) Vol. 19, No. 3, pp. 211-219. .
Refs: 38
ISSN: 0282-0080 CODEN: GLJOEW
CY Netherlands
DT Journal; Article
FS 026 Immunology, Serology and Transplantation
029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 31 Jul 2003
Last Updated on STN: 31 Jul 2003
AB The bisecting GlcNAc is transferred to complex or hybrid N-glycans by the action of N-. ***acetylglucosaminyltransferase*** ***III*** (GlcNAc-TIII) encoded by the Mgat3 gene. CHO ***cells*** expressing mouse GlcNAc-TIII were shown by matrix-assisted laser desorption ionization (MALDI) mass spectrometry to produce mainly complex N-glycans with the predicted extra (bisecting) GlcNAc. In order to probe biological functions of the bisecting GlcNAc, antibodies that recognize this residue in the context of complex ***cell*** surface glycoconjugates were sought. The LEC10 gain-of-function Chinese hamster ovary (CHO) ***cell*** mutant that expresses GlcNAc-TIII and complex N-glycans with the bisecting GlcNAc was used to immunize Mgat3(+/+) and Mgat3(-/-) mice. ELISA of whole sera showed that polyclonal antibodies that bound specifically to LEC10 ***cells*** were obtained solely from Mgat3(-/-) mice. Fluorescence-activated ***cell*** cytometry of different CHO glycosylation mutants and western blotting after glycosidase treatments were used to show that anti-LEC10 ***cell*** antisera from Mgat3(-/-) mice recognize ***cellular*** glycoproteins with complex N-glycans

containing both a bisecting GlcNAc and Gal residues. The polyclonal antibody specificity was similar to that of the lectin E-PHA. IgM-depleted serum containing IgG and IgA antibodies retained full binding activity. Therefore Mgat3(-/-) mice but not wild type mice can be used effectively to produce polyclonal antibodies that specifically recognize glycoproteins bearing complex N-glycans with a bisecting GlcNAc.

L5 ANSWER 11 OF 206 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2003288080 EMBASE <<LOGINID::20070409>>
TI Two closely related forms of UDP-GlcNAc: .alpha.6-D-mannoside .beta.1,2-N-acetylglucosaminyl-transferase II occur in the clawed frog Xenopus laevis.
AU Mucha J.; Svoboda B.; Kappel S.; Strasser R.; Bencur P.; Frohwein U.; Schachter H.; Mach L.; Glossl J.
CS L. Mach, Zentrum fur Angewandte Genetik, Univ. fur Bodenkultur Wien, Muthgasse 18, A-1190 Wien, Austria. lukas.mach@boku.ac.at
SO Glycoconjugate Journal, (1 Mar 2003) Vol. 19, No. 3, pp. 187-195. .
Refs: 31
ISSN: 0282-0080 CODEN: GLJOEW
CY Netherlands
DT Journal; Article
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 31 Jul 2003
Last Updated on STN: 31 Jul 2003
AB UDP-GlcNAc:.alpha.6-D-mannoside .beta.1,2-N-***acetylglucosaminyltransferase*** ***II*** (GnT ***II*** ; EC 2.4.1.143) is a medial-Golgi resident enzyme that catalyses an essential step in the biosynthetic pathway leading from high mannose to complex N-linked oligosaccharides. Screening a cDNA library from Xenopus laevis ovary with a human GnT II DNA probe resulted in the isolation of two cDNA clones encoding two closely related GnT II isoenzymes, GnT II-A and GnT II-B. Analysis of the corresponding genomic DNAs revealed that the open reading frame of both X. laevis GnT II genes resides within a single exon. The GnT II-A gene was found to be transcriptionally active in all X. laevis tissues tested. In contrast, expression of the GnT II-B gene was detected only in a limited number of tissues. Both GnT II-A and GnT II-B exhibit a type II transmembrane protein topology with a putative N-terminal cytoplasmic tail of 9 amino acids followed by a transmembrane domain of 18 residues, and a C-terminal luminal domain of 405 residues. The two proteins differ at 28 amino acid positions within their luminal regions. Heterologous expression of soluble forms of the enzymes in insect ***cells*** showed that GnT II-A and GnT II-B are both catalytically active and exhibit similar specific activities. Both recombinant proteins are modified with N-linked oligosaccharides. N-terminal deletion studies demonstrated that the first 49 amino acid residues are not essential for proper folding and enzymatic activity of X. laevis GnT II.

L5 ANSWER 12 OF 206 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2003283327 EMBASE <<LOGINID::20070409>>
TI Caveolin-1 regulates the functional localization of N-***acetylglucosaminyltransferase*** ***III*** within the Golgi apparatus.
AU Sasai K.; Ikeda Y.; Ihara H.; Honke K.; Taniguchi N.
CS United States. proftani@biochem.med.osaka-u.ac.jp
SO Journal of Biological Chemistry, (11 Jul 2003) Vol. 278, No. 28, pp. 25295-25301. .
Refs: 36
ISSN: 0021-9258 CODEN: JBCHA3
CY United States
DT Journal; Article
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 31 Jul 2003
Last Updated on STN: 31 Jul 2003
AB In an investigation of the mechanism underlying the functional sublocalization of glycosyltransferases within the Golgi apparatus, caveolin-1 was identified as a possible ***cellular*** factor. Caveolin-1 appears to regulate the localization of N-***acetylglucosaminyltransferase*** ***III*** (GnT- ***III***) in the intra-Golgi subcompartment. Structural analyses of total ***cellular*** N-glycans indicated that the overexpression of GnT-III in human hepatoma ***cells*** , in which caveolin-1 is not expressed, failed to reduce branch formation, whereas expression of caveolin-1 led to a dramatic decrease in the extent of branching with no enhancement in GnT-III activity. Because the addition of a bisecting GlcNAc by GnT-III to the core .beta.-Man in N-glycans prevents the action of GnT-IV and GnT-V, both of which are involved in branch formation, this result suggests that caveolin-1 facilitates the prior action of GnT-III, relative to the other GnTs, on the nascent sugar chains in the Golgi apparatus and that GnT-III is redistributed in the earlier Golgi subcompartment by caveolin-1. Indeed, when caveolin-1 was expressed in human hepatoma ***cells*** , it was found to be co-localized with GnT-III, as evidenced by the fractionation of Triton X-100-insoluble ***cellular*** membranes by density gradient ultracentrifugation. Caveolin-1 may modify the biosynthetic pathway of sugar chains via the regulation of the intra-Golgi subcompartment localization of this key glycosyltransferase.

L5 ANSWER 13 OF 206 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2003270111 EMBASE <<LOGINID::20070409>>
TI Down-regulation of hydrogen peroxide-induced PKC.delta. activation in N-***acetylglucosaminyltransferase*** ***III*** -transfected HeLaS3 ***cells*** .
AU Shibukawa Y.; Takahashi M.; Laffont I.; Honke K.; Taniguchi N.
CS N. Taniguchi, Department of Biochemistry, Osaka Univ. Grad. School of Medicine, 2-2 Yamadaoka, Suita, Osaka 565-0871, Japan. proftani@biochem.med.osaka-u.ac.jp
SO Journal of Biological Chemistry, (31 Jan 2003) Vol. 278, No. 5, pp. 3197-3203. .
Refs: 37
ISSN: 0021-9258 CODEN: JBCHA3
CY United States
DT Journal; Article
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 24 Jul 2003
Last Updated on STN: 24 Jul 2003
AB N- ***acetylglucosaminyltransferase*** ***III*** (GnT- ***III***) is a key enzyme that inhibits the extension of N-glycans by introducing a bisecting N-acetylglucosamine residue. Our previous studies have shown that modification of N-glycans by GnT-III affects a number of intracellular signaling pathways. In this study, the effects of GnT-III on the ***cellular*** response to reactive oxygen species (ROS) were examined. We found that an overexpression of GnT-III suppresses H(2)O(2)-induced apoptosis in HeLaS3 ***cells*** . In the case of GnT-III transfectants, activation of Jun N-terminal kinase (JNK) following H(2)O(2) treatment was markedly reduced compared with control ***cells*** . Either the depletion of protein kinase C (PKC) by prolonged treatment with phorbol 12-myristate 13-acetate or the inhibition of PKC by the specific inhibitor H7 attenuated the H(2)O(2)-induced activation of JNK1 and apoptosis in control ***cells*** but not in the GnT-III transfectants. Furthermore, we found that H(2)O(2)-induced phosphorylation of PKC.delta. was markedly suppressed in GnT-III transfectants. Rottlerin, a specific inhibitor of PKC.delta., significantly inhibited H(2)O(2)-induced activation of JNK1 in control ***cells*** , indicating that PKC.delta. is involved in the pathway. These findings suggest that the overexpression of GnT-III suppresses H(2)O(2)-induced activation of PKC.delta.-JNK1 pathway, resulting in inhibition of apoptosis.

L5 ANSWER 14 OF 206 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2003247774 EMBASE <<LOGINID::20070409>>
TI Co-effect of HLA-G1 and glycosyltransferases in reducing NK ***cell*** -mediated pig endothelial ***cell*** lysis.
AU Miyagawa S.; Nakai R.; Matsunami K.; Kusama T.; Shirakura R.
CS S. Miyagawa, Division of Organ Transplantation, Department of Regenerative Medicine, Osaka University, 2-2 Yamadaoka, Suita, Osaka 565-0871, Japan. miyagawa@orgtrp.med.osaka-u.ac.jp
SO Transplant Immunology, (2003) Vol. 11, No. 2, pp. 147-153. .
Refs: 35
ISSN: 0966-3274 CODEN: TRIME2
PUI S 0966-3274(02)00151-X
CY United Kingdom
DT Journal; Article
FS 026 Immunology, Serology and Transplantation
029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 3 Jul 2003
Last Updated on STN: 3 Jul 2003
AB Natural killer (NK) ***cells*** play an important role in xenograft rejection. The aim of this study was to evaluate the co-effect of human leukocyte antigen (HLA)-G1 expression and the remodeling of glycoantigens such as the .alpha.-Gal epitope, Ga1.alpha.1,3Ga1.beta.1,4GlcNAc-R, by the introduction of glycosyltransferase genes related to NK ***cell*** -mediated direct cytotoxicity. Human peripheral blood mononuclear ***cells*** or an NK-like ***cell*** line, YT ***cells*** , was used as an effector and pig endothelial ***cells*** (PEC) as the target. A PEC transfectant with HLA-G1 was first prepared by the transfection of HLA-G1 and human .beta.2 microglobulin. Several new transfectants were then established by the transfection of glycosyltransferase to the HLA-G1 transfectant. The effect of HLA-G1 on NK ***cell*** -mediated PEC lysis was lower than that by the glycosyltransferases. Therefore, in the case of the co-transfectants except for HLA-G1 + .alpha.2,6sialyltransferase, such as HLA-G1 + N-***acetylglucosaminyltransferase*** - ***III*** and HLA-G1 + .alpha.1,2fucosyltransferase, the effect of HLA-G1 expression on NK-mediated killing appeared to be accounted for by the transfected glycosyltransferase activities and the reduced .alpha.-Gal expression on the ***cell*** surface. However, these transfectants showed significant reductions in direct NK ***cell*** -mediated cytotoxicity, compared with the single HLA-G1 transfectant. The results herein suggest that a combination of HLA-G1 and glycosyltransferases has considerable potential for the downregulation of NK ***cell*** -mediated cytolysis.

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AN 2003135027 EMBASE <<LOGINID::20070409>>
TI Complex-type biantennary N-glycans of recombinant human transferrin from Trichoplusia ni insect ***cells*** expressing mammalian .beta.-1,4-galactosyltransferase and .beta.-1,2-N-***acetylglucosaminyltransferase*** ***II*** .
AU Tomiya N.; Howe D.; Aumiller J.J.; Pathak M.; Park J.; Palter K.B.; Jarvis D.L.; Betenbaugh M.J.; Lee Y.C.
CS N. Tomiya, Department of Biology, Johns Hopkins University, 3400 North Charles Street, Baltimore, MD 21218, United States. ntomiya1@jhu.edu
SO Glycobiology, (1 Jan 2003) Vol. 13, No. 1, pp. 23-34. .
Refs: 47
ISSN: 0959-6658 CODEN: GLYCE3
CY United Kingdom
DT Journal; Article
FS 004 Microbiology
LA English
SL English
ED Entered STN: 17 Apr 2003
Last Updated on STN: 17 Apr 2003
AB A novel recombinant baculovirus expression vector was used to produce His-tagged human transferrin in a transformed insect ***cell*** line (Tn5.beta.4GalT) that constitutively expresses a mammalian .beta.-1,4-galactosyltransferase. This virus encoded the His-tagged human transferrin protein in conventional fashion under the control of the very late polyhedrin promoter. In addition, to enhance the synthesis of galactosylated biantennary N-glycans, this virus encoded human .beta.-1,2-N- ***acetylglucosaminyltransferase*** ***II*** under the control of an immediate-early (ie1) promoter. Detailed analyses by MALDI-TOF MS, exoglycosidase digestion, and two-dimensional HPLC revealed
that the N-glycans on the purified recombinant human transferrin produced by this virus-host system included four different fully galactosylated, biantennary, complex-type glycans. Thus, this study describes a novel baculovirus-host system, which can be used to produce a recombinant glycoprotein with fully galactosylated, biantennary N-glycans.

L5 ANSWER 16 OF 206 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2003074848 EMBASE <<LOGINID::20070409>>
TI Transgenic pigs expressing both human decay-accelerating factor and N-***acetylglucosaminyltransferase*** ***III*** .
AU Takahagi Y.; Miyagawa S.; Murakami H.; Matsunami K.; Fujimura T.; Shigehisa T.; Shirakura R.
CS Y. Takahagi, Anim. Engineering Research Institute, 3-3 Midorigahara, Tsukuba, Ibaraki 300-2646, Japan. takahagi@rdc.nipponham.co.jp
SO Transplantation Proceedings, (2003) Vol. 35, No. 1, pp. 516-517. .
Refs: 4
ISSN: 0041-1345 CODEN: TRPPA8
CY United States
DT Journal; Conference Article
FS 009 Surgery
026 Immunology, Serology and Transplantation
029 Clinical Biochemistry
LA English
ED Entered STN: 27 Feb 2003
Last Updated on STN: 27 Feb 2003
DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

L5 ANSWER 17 OF 206 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2002458926 EMBASE <<LOGINID::20070409>>
TI Engineering the protein N-glycosylation pathway in insect ***cells*** for production of biantennary, complex N-glycans.
AU Hollister J.; Grabenhorst E.; Nimtz M.; Conradt H.; Jarvis D.L.
CS D.L. Jarvis, Department of Molecular Biology, University of Wyoming, Laramie, WY 82071, United States. dljarvis@uwyo.edu
SO Biochemistry, (17 Dec 2002) Vol. 41, No. 50, pp. 15093-15104. .
Refs: 47
ISSN: 0006-2960 CODEN: BICHAW
CY United States
DT Journal; Article
FS 004 Microbiology
LA English
SL English
ED Entered STN: 9 Jan 2003
Last Updated on STN: 9 Jan 2003
AB Insect ***cells*** , like other eucaryotic ***cells*** , modify many of their proteins by N-glycosylation. However, the endogenous insect ***cell*** N-glycan processing machinery generally does not produce complex, terminally sialylated N-glycans such as those found in mammalian systems. This difference in the N-glycan processing pathways of insect ***cells*** and higher eucaryotes imposes a significant limitation on their use as hosts for baculovirus-mediated recombinant glycoprotein production. To address this problem, we previously isolated two transgenic insect ***cell*** lines that have mammalian .beta.1,4-galactosyltransferase or .beta.1,4-galactosyltransferase and .alpha.2,6-sialyltransferase genes. Unlike the parental insect ***cell*** line, both transgenic ***cell*** lines expressed the mammalian glycosyltransferases and were able to produce terminally galactosylated or sialylated N-glycans. The purpose of the present study was to investigate the structures of the N-glycans produced by these transgenic insect ***cell*** lines in further detail. Direct structural analyses revealed that the most extensively processed N-glycans produced by the transgenic insect ***cell*** lines were novel, monoantennary structures with elongation of only the .alpha.1,3 branch. This led to the hypothesis that the transgenic insect ***cell*** lines lacked adequate endogenous N- ***acetylglucosaminyltransferase*** ***II*** activity for biantennary N-glycan production. To test this hypothesis and further extend the N-glycan processing pathway in Sf9 ***cells*** , we produced a new transgenic line designed to constitutively express a more complete array of mammalian glycosyltransferases, including N- ***acetylglucosaminyltransferase*** ***II*** . This new transgenic insect ***cell*** line, designated SfSWT-1, has higher levels of five glycosyltransferase activities than the parental ***cells*** and supports baculovirus replication at normal levels. In addition, direct structural analyses showed that SfSWT-1 ***cells*** could produce biantennary, terminally sialylated N-glycans. Thus, this study provides new insight on the glycobiology of insect ***cells*** and describes a new transgenic insect ***cell*** line that will be widely useful for the production of more authentic recombinant glycoproteins by baculovirus expression vectors.

L5 ANSWER 18 OF 206 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2002416434 EMBASE <<LOGINID::20070409>>
TI Biological consequences of overexpressing or eliminating N-acetylglucosaminyltransferase-TIII in the mouse.
AU Stanley P.
CS P. Stanley, Department of Cell Biology, Albert Einstein College Medicine, Yeshiva University, 1300 Morris Park Avenue, Bronx, NY 10461, United States. stanley@aecom.yu.edu
SO Biochimica et Biophysica Acta - General Subjects, (19 Dec 2002) Vol. 1573, No. 3, pp. 363-368. .
Refs: 39
ISSN: 0304-4165 CODEN: BBGSB3
PUI S 0304-4165(02)00404-X
CY Netherlands
DT Journal; General Review
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 5 Dec 2002
Last Updated on STN: 5 Dec 2002
AB N- ***acetylglucosaminyltransferase*** ***III*** (GlcNAc-TIII), a product of the human MGAT3 gene, was discovered as a glycosyltransferase activity in hen oviduct. GlcNAc-TIII transfers GlcNAc in .beta.4-linkage to the core Man of complex or hybrid N-glycans, and thereby alters not only the composition, but also the conformation of the N-glycan. The dramatic consequences of the addition of this bisecting GlcNAc residue are reflected in the altered binding of lectins that recognize Gal residues on N-glycans. Changes in GlcNAc-TIII expression correlate with hepatoma and leukemia in rodents and humans, and the bisecting GlcNAc on Asn 297 of human IgG antibodies enhances their effector functions. Overexpression of a cDNA encoding GlcNAc-TIII alters growth control and ***cell*** - ***cell*** interactions in cultured ***cells*** , and in transgenic mice. While mice lacking GlcNAc-TIII are viable and fertile, they exhibit retarded progression of diethylnitrosamine (DEN)-induced liver tumors. Further biological functions of GlcNAc-TIII are expected to be uncovered as mice with a null mutation in the Mgat3 gene are challenged. .COPYRGT. 2002 Elsevier Science B.V. All rights reserved.

L5 ANSWER 19 OF 206 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2002416428 EMBASE <<LOGINID::20070409>>
TI Mice with a homozygous deletion of the Mgat2 gene encoding UDP-N-acetylglucosamine:.alpha.-6-D-mannoside .beta.1,2-N-***acetylglucosaminyltransferase*** ***II*** : A model for congenital disorder of glycosylation type IIa.
AU Wang Y.; Schachter H.; Marth J.D.
CS H. Schachter, Hospital for Sick Children, Department of Biochemistry, University of Toronto, 555 University Avenue, Toronto, Ont. M5G 1X8, Canada. harry@sickkids.ON.CA
SO Biochimica et Biophysica Acta - General Subjects, (19 Dec 2002) Vol. 1573, No. 3, pp. 301-311. .
Refs: 36
ISSN: 0304-4165 CODEN: BBGSB3
PUI S 0304-4165(02)00397-5
CY Netherlands
DT Journal; General Review
FS 005 General Pathology and Pathological Anatomy
021 Developmental Biology and Teratology
022 Human Genetics
029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 5 Dec 2002
Last Updated on STN: 5 Dec 2002
AB Mice homozygous for a deletion of the Mgat2 gene encoding UDP-N-acetylglucosamine:.alpha.-6-D-mannoside .beta.1,2-N-***acetylglucosaminyltransferase*** ***II*** (GlcNAcT- ***II*** , EC 2.4.1.143) have been reported. GlcNAcT-II is essential for the synthesis of complex N-glycans. The Mgat2-null mice were studied in a comparison with the symptoms of congenital disorder of glycosylation type IIa (CDG-IIa) in humans. Mutant mouse tissues were shown to be deficient in GlcNAcT-II enzyme activity and complex N-glycan synthesis, resulting in severe gastrointestinal, hematologic and osteogenic abnormalities. All

mutant mice died in early post-natal development. However, crossing the Mgat2 mutation into a distinct genetic background resulted in a low frequency of survivors exhibiting additional and novel disease signs of CDG-IIa. Analysis of N-glycan structures in the kidneys of Mgat2-null mice showed a novel bisected hybrid N-glycan structure in which the bisecting GlcNAc residue was substituted with a .beta.1,4-linked galactose or the Lewis(x) structure. These studies suggest that some of the functions of complex N-glycan branches are conserved in mammals and that human disease due to aberrant protein N-glycosylation may be modeled in the mouse, with the expectation in this case of gaining insights into CDG-IIa disease pathogenesis. Further analyses of the Mgat2-deficient phenotype in the mouse have been accomplished involving ***cells*** in which the Mgat2 gene is dispensable, as well as other ***cell*** lineages in which a severe defect is present. Pre-natal defects appear in a significant number of embryos, and likely reflect a limited window of time in which a future therapeutic approach might effectively operate.

L5 ANSWER 20 OF 206 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2002312012 EMBASE <<LOGINID::20070409>>
TI Truncated, inactive N- ***acetylglucosaminyltransferase*** ***III*** (GlcNAc-TIII) induces neurological and other traits absent in mice that lack GlcNAc-TIII.
AU Bhattacharyya R.; Bhaumik M.; Raju T.S.; Stanley P.
CS P. Stanley, Dept. of Cell Biology, Albert Einstein College of Medicine, 1300 Morris Park Ave., New York, NY 10461, United States. stanley@aecom.yu.edu
SO Journal of Biological Chemistry, (19 Jul 2002) Vol. 277, No. 29, pp. 26300-26309. .
Refs: 52
ISSN: 0021-9258 CODEN: JBCHA3
CY United States
DT Journal; Article
FS 008 Neurology and Neurosurgery
021 Developmental Biology and Teratology
022 Human Genetics
029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 19 Sep 2002
Last Updated on STN: 19 Sep 2002
AB N- ***Acetylglucosaminyltransferase*** ***III*** (GlcNAc-TIII), the product of the Mgat3 gene, transfers the bisecting GlcNAc to the core mannose of complex N-glycans. The addition of this residue is regulated during development and has functional consequences for receptor signaling, ***cell*** adhesion, and tumor progression. Mice homozygous for a null mutation at the Mgat3 locus (Mgat3(.DELTA.)) or for a targeted mutation in the Mgat3 gene (previously called Mgat3(neo), but herein renamed Mgat3(T37) because the allele generates inactive GlcNAc-TIII of .apprx.37 kDa) were found to exhibit retarded progression of liver tumors. Matrix-assisted laser desorption/ionization time-of-flight mass spectrometry of neutral N-glycans from kidneys revealed no significant differences, and both mutants showed the expected lack of N-glycan species with an additional GlcNAc. However, the two mutants differed in several biological traits. Mgat3(T37/T37) homozygotes in a mixed or 129(SvJ) background were retarded in growth rate and exhibited an altered leg clasp reflex, an altered gait, and defective nursing behavior. Pups abandoned by Mgat3(T37/T37) mothers were rescued by wild-type foster mothers. None of these Mgat3(T37/T37) traits were exhibited by Mgat3(.DELTA./.DELTA.) mice or by heterozygous mice carrying the Mgat3(T37) mutation. Similarly, no dominant-negative effect was observed in Chinese hamster ovary ***cells*** expressing truncated GlcNAc-TIII in the presence of wild-type GlcNAc-TIII. However, compound heterozygotes carrying both the Mgat3(T37) and Mgat3.DELTA. mutations exhibited a marked leg clasp reflex, indicating that in the absence of wild-type GlcNAc-TIII, truncated GlcNAc-TIII causes this phenotype. The Mgat3 gene was expressed in brain at embryonic day 10.5 and thereafter and in neurons of adult cerebellum. The mutant Mgat3 gene was also highly expressed in Mgat3(T37/T37) brain. This may be the basis of the unexpected neurological phenotype induced by truncated, inactive GlcNAc-TIII in the mouse.

L5 ANSWER 21 OF 206 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2002046495 EMBASE <<LOGINID::20070409>>
TI A catalytically inactive .beta.1,4-N- ***acetylglucosaminyltransferase*** ***III*** (GnT- ***III***) behaves as a dominant negative GnT-III inhibitor.
AU Ihara H.; Ikeda Y.; Koyota S.; Endo T.; Honke K.; Taniguchi N.
CS N. Taniguchi, Department of Biochemistry, Osaka University Medical School, 2-2 Yamadaoka, Suita, Osaka 565-0871, Japan. proftani@biochem.med.osaka-u.ac.jp
SO European Journal of Biochemistry, (2002) Vol. 269, No. 1, pp. 193-201. .
Refs: 45
ISSN: 0014-2956 CODEN: EJBCAI
CY United Kingdom
DT Journal; Article
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 14 Feb 2002
Last Updated on STN: 14 Feb 2002
AB .beta.1,4-N- ***Acetylglucosaminyltransferase*** ***III*** (GnT-***III***) plays a regulatory role in the biosynthesis of N-glycans, and it has been suggested that its product, a bisecting GlcNAc, is involved in a variety of biological events as well as in regulating the biosynthesis of the oligosaccharides. In this study, it was found, on the basis of sequence homology, that GnT-III contains a small region that is significantly homologous to both snail .beta.1,4GlcNAc transferase and .beta.1,4Gal transferase-1. Subsequent mutational analysis demonstrated an absolute requirement for two conserved Asp residues (Asp321 and Asp323), which are located in the most homologous region of rat GnT-III, for enzymatic activity. The overexpression of Asp323-substituted, catalytically inactive GnT-III in Huh6 ***cells*** led to the suppression of the activity of endogenous GnT-III, but no significant decrease in its expression, and led to a specific inhibition of the formation of bisected sugar chains, as shown by structural analysis of the total N-glycans from the ***cells*** . These findings indicate that the mutant serves a dominant negative effect on a specific step in N-glycan biosynthesis. This type of 'dominant negative glycosyltransferase', identified has potential value as a powerful tool for defining the precise biological roles of the bisecting GlcNAc structure.

L5 ANSWER 22 OF 206 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2001440906 EMBASE <<LOGINID::20070409>>
TI Expression of bisecting N- ***acetylglucosaminyltransferase*** - ***III*** in human hepatocarcinoma tissues, fetal liver tissues, and hepatoma ***cell*** lines of Hep3B and HepG2.
AU Song E.-Y.; Kang S.-K.; Lee Y.-C.; Park Y.-G.; Chung T.-H.; Kwon D.-H.; Byun S.-M.; Kim C.-H.
CS Dr. C.-H. Kim, Department of Biochemistry, College of Oriental Medicine, Dongguk University, Sukjang-Dong 707, Kyungju 780-714, Korea, Republic of. chkimbio@mail.dongguk.ac.kr
SO Cancer Investigation, (2001) Vol. 19, No. 8, pp. 799-807. .
Refs: 29
ISSN: 0735-7907 CODEN: CINVD7
CY United States
DT Journal; Article
FS 016 Cancer
029 Clinical Biochemistry
048 Gastroenterology
LA English
SL English
ED Entered STN: 10 Jan 2002
Last Updated on STN: 10 Jan 2002
AB In this paper, uridine diphosphate (UDP)-N-acetylglucosamine/.beta.-D-mannoside .beta.-1,4 N- ***acetylglucosaminyltransferase*** ***III*** (GlcNAc-transferase- ***III*** C 2.4.1.144) activity was determined in human hepatoma ***cell*** lines of Hep3B and HepG2, and also compared with those of normal liver tissues and primary hepatocytes. GlcNAc-transferase-III enzymes of Hep3B and HepG2 were mainly detected in the membrane fraction. When GlcN, GlcN-biant-PA and UDP-GlcNAc were used as substrates, the K(m) values (4.7 mM for UDP-GlcNAc and 1.1 mM for GlcN, GlcN-biant-PA) of Hep3B GlcNAc-transferase-III were distinguishable from those of HepG2 GlcNAc-transferase-III (6.8 mM for UDP-GlcNAc and 3.4 mM for GlcN, GlcN-biant-PA). Furthermore, Hep3B enzyme in membrane fraction showed about 1.5-fold higher specific activity (1423 pmol/hr/mg) than that of HepG2 (1066 pmol/hr/mg). Normal liver ***cells*** and primary adult hepatocytes are characterized by a very low level of GlcNAc-transferase-III activity, whereas human hepatoma ***cells*** exhibited high activities. These data were supported by reverse transcription-polymerase chain reaction results, showing that expression of the GlcNAc-transferase-III mRNA increased in proportion to the enzymatic activities. Although the mechanism underlying the induction of this enzyme is unknown, lectin blot analysis showed that oligosaccharides in many glycoproteins were observed in hepatoma ***cells*** . By treating hepatocarcinoma cultures that express GlcNAc-transferase-III with inhibitors (tunicamycin, deoxymannojirimycin, and swainsonine) of different steps of the glycosylation, we provide evidence that expression of GlcNAc-transferase-III mRNA is dependent on glycosylation of ***cellular*** proteins.

L5 ANSWER 23 OF 206 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2001383586 EMBASE <<LOGINID::20070409>>
TI mRNA expression of three glycosyltransferases in human hepatoma tissues.
AU Chen G.; Guan M.; Su B.; Lu Y.
CS Y. Lu, Department of Laboratory Medicine, Hua Shan Hospital, Fudan University, 12 Wulumuqi Zhong Road, Shanghai 200040, China. yuanlu8@public7.sta.net.cn
SO Clinica Chimica Acta, (2001) Vol. 313, No. 1-2, pp. 77-80. .
Refs: 13
ISSN: 0009-8981 CODEN: CCATAR
PUI S 0009-8981(01)00652-0
CY Netherlands
DT Journal; Conference Article
FS 016 Cancer
029 Clinical Biochemistry
048 Gastroenterology
LA English
SL English
ED Entered STN: 15 Nov 2001
Last Updated on STN: 15 Nov 2001

AB Background: The sugar-chain structures of many glycoproteins are altered in the hepatoma tissues. The molecular mechanism by which these alterations occur remains largely unknown. Methods: Messeger RNA expression of N-acetylglucosaminyltransferase (GlcNAcT-V), N-***acetylglucosaminyltransferase*** ***III*** (GlcNAcT- ***III***) and .alpha.1-6 fucosyltransferase (.alpha.1-6 FucT), were investigated in normal liver tissues samples (n = 7), primary hepatic cancer (PHC) tissues (n = 15) and noncancerous tissues surrounding PHC (n = 15) by reverse transcriptase-polymerase chain reaction (RT-PCR). Results: The mRNA expression of the three glycosyltransferases in PHC tissues were enhanced by 3- to 10-fold in comparison with that in normal liver tissues. There were significantly higher mRNA expressions of GlcNAcT-V in invasive PHC than non-invasive. Conclusions: The change of glycoprotein sugar-chain structures in PHC may be related to the abnormal mRNA expression of some glycosyltransferases and the levels of mRNA expression of GlcNAcT-V associated with PHC invasiveness. .COPYRGT. 2001 Elsevier Science B.V. All rights reserved.

L5 ANSWER 24 OF 206 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2001335998 EMBASE <<LOGINID::20070409>>
TI Congenital disorders of glycosylation type Ia and IIa are associated with different primary haemostatic complications.
AU Van Geet C.; Jaeken J.; Freson K.; Lenaerts T.; Arnout J.; Vermylen J.; Hoylaerts M.F.
CS C. Van Geet, Department of Paediatrics, UZ Gasthuisberg, University of Leuven, Herestraat 49, 3000 Leuven, Belgium. Christel.Vangeet@uz.kuleuven.ac.be
SO Journal of Inherited Metabolic Disease, (2001) Vol. 24, No. 4, pp. 477-492. .
Refs: 18
ISSN: 0141-8955 CODEN: JIMDDP
CY Netherlands
DT Journal; Article
FS 025 Hematology
029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 11 Oct 2001
Last Updated on STN: 11 Oct 2001
AB Congenital disorders of glycosylation (CDG) type I are mostly due to a deficient phosphomannomutase activity, called CDG Ia. CDG IIa (mutations in the MGAT2 gene) results from a deficient activity of the Golgi enzyme N- ***acetylglucosaminyltransferase*** ***II*** . CDG Ia patients predominantly have a thrombotic tendency, whereas our CDG IIa patient has an increased bleeding tendency, despite similar coagulation factor abnormalities in both types. We have investigated whether abnormally glycosylated platelet membrane glycoproteins are involved in the haemostatic complications of both CDG groups. In flow cytometry, the binding of Ricinus communis lectin (reactive with .beta.-galactose primarily) to control platelets increased after neuraminidase treatment: this increase was smaller ($p < 0.01$) in CDG Ia patients (3.1 .+-. 0.08 times) than in control platelets (8.5 .+-. 1.8 times) and did not occur in the CDG IIa patient. Platelet-rich plasma from CDG Ia patients, but not a CDG IIa patient, aggregated spontaneously and gel-filtered platelets from CDG Ia patients agglutinated at very low concentrations of ristocetin, independently of von Willebrand factor (vWF). Accordingly, in stirred whole blood, the rate of single platelet disappearance of CDG Ia patients was twice that of control platelets. In contrast, perfusion of whole anticoagulated blood of the CDG IIa patient over collagen yielded markedly decreased platelet adherence to collagen at shear rates involving glycoprotein (GP) Ib-vWF interactions. Thus, abnormal glycosylation of platelet glycoproteins in CDG Ia enhances nonspecific platelet interactions, in agreement with a thrombotic tendency. The reduced GP Ib-mediated platelet reactivity with vessel wall components in the CDG IIa patient under flow conditions provides a basis for his bleeding tendency.

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AN 2001264683 EMBASE <<LOGINID::20070409>>
TI Overexpression of sialyltransferase CMP-sialic acid: Gal.beta.1,3GalNAc-R .alpha.6-sialyltransferase is related to poor patient survival in human colorectal carcinomas.
AU Schneider F.; Kemmner W.; Haensch W.; Franke G.; Gretschel S.; Karsten U.; Schlag P.M.
CS W. Kemmner, Robert-Rossle-Klinik, Max Delbruck Ctr. for Molec. Med., Department for Surgery, Lindenberger Weg 80, D-13122 Berlin, Germany. wkemmner@mdc-berlin.de
SO Cancer Research, (1 Jun 2001) Vol. 61, No. 11, pp. 4605-4611. .
Refs: 23
ISSN: 0008-5472 CODEN: CNREA8
CY United States
DT Journal; Article
FS 016 Cancer
048 Gastroenterology
LA English
SL English
ED Entered STN: 23 Aug 2001
Last Updated on STN: 23 Aug 2001
AB Thomsen-Friedenreich (TF)-related blood group antigens, such as TF, Tn, and their sialylated variants, belong to a family of tumor-associated carbohydrates. The aim of the present study was to examine tumor-associated alterations of glycosyltransferases involved in the biosynthesis of the TF glycotope in colorectal carcinomas. To this end, glycosyltransferase expression was examined in 40 cases of colorectal carcinoma specimens classified according to the WHO/Union International Centre Cancer guidelines and in "normal" mucosa of the same patients. Occurrence of TF glycotope was examined by immunohistochemistry with the monoclonal antibody A78-G/A7. Expression of sialyltransferases CMP-sialic acid:Gal.beta.1,3GalNAc-R .alpha.3-sialyltransferase I and II (ST3Gal-I and ST3Gal-II) and CMP-sialic acid: Gal.beta.1,3GalNAc-R .alpha.6-sialyltransferase (ST6GalNAc-II) and of core 2 .beta.1,6-N-acetylglucosaminyltransferase was determined by reverse transcription-PCR in the same cryostat sections used for immunohistochemistry. Additionally, .alpha.2,3-sialyltransferase enzyme activity was studied in each of these tissues. The TF glycotope was detected in 7% of the normal mucosa, but in 57% of the carcinoma samples. Expression of .alpha.2,3-sialyltransferases ST3Gal-I, ST3Gal-II, and enzyme activity of .alpha.2,3-sialyltransferase was significantly increased ($P < 0.001$) in carcinoma specimens compared with normal mucosa. ST3Gal-I mRNA expression was significantly increased ($P = 0.05$) in cases showing invasion of lymph vessels. Expression of ST6GalNAc-II was significantly increased ($P = 0.04$) in cases with metastases to lymph nodes along the vascular trunk. Moreover, ST6GalNAc-II expression provides an prognostic factor for patient survival (log rank, $P = 0.02$). In an attempt to study the functional relevance of the glycosyltransferases for TF biosynthesis, SW480 colorectal ***cells*** were transfected with each of the enzymes, and ***cell*** surface expression of the TF glycotope was examined by flow cytometry. The presence of TF was not altered by transfection of the ***cells*** with either sialyltransferase ST3Gal-I or ST3Gal-II. However, successful transfection with core 2 .beta.1,6-N-acetylglucosaminyltransferase led to reduced expression of TF. In contrast, increased ***cell*** surface expression of TF was found after ST6GalNAc-II transfection. Thus, expression of TF on the ***cell*** surface of SW480 colorectal carcinoma ***cells*** depends on the ratio of core 2 .beta.1,6-N-***acetylglucosaminyltransferase*** and ST6GalNAc- ***II*** . Earlier immunohistological studies demonstrated that TF is a prognostic factor for patient survival. Our results suggest that sialyltransferase ST6GalNAc-II is of crucial relevance for the prognostic significance of TF.

L5 ANSWER 26 OF 206 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2001261192 EMBASE <<LOGINID::20070409>>
TI Expression of GnTIII in a recombinant anti-CD20 CHO production ***cell*** line: Expression of antibodies with altered glycoforms leads to an increase in ADCC through higher affinity for Fc.gamma.RIII.
AU Davies J.; Jiang L.; Pan L.-Z.; LaBarre M.J.; Anderson D.; Reff M.
CS M. Reff, IDEC Pharmaceuticals Corporation, 3010 Science Park Road, San Diego, CA 92191-9080, United States. mreff@idecpharm.com
SO Biotechnology and Bioengineering, (20 Aug 2001) Vol. 74, No. 4, pp. 288-294. .
Refs: 15
ISSN: 0006-3592 CODEN: BIBIAU
CY United States
DT Journal; Article
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 15 Aug 2001
Last Updated on STN: 15 Aug 2001
AB The gene encoding the rat glycosylation enzyme .beta.1-4-N-***acetylglucosaminyltransferase*** ***III*** (GnTIII) was cloned and coexpressed in a recombinant production Chinese hamster ovary (CHO) ***cell*** line expressing a chimeric mouse/human anti-CD20 IgG1 antibody. The new ***cell*** lines expressed high levels of antibody and have growth kinetics similar to that of the parent. Relative QPCR showed the ***cell*** lines to express varying levels of mRNA. High-performance liquid chromatography (HPLC) analysis showed the enzyme to have added bisecting N-acetylglucosamine (GlcNAc) residues in most (48% to 71%) of the N-linked oligosaccharides isolated from antibody preparations purified from the ***cell*** lines. In an ADCC assay the new antibody preparations promoted killing of CD20-positive target ***cells*** at approximately 10- to 20-fold lower concentrations than the parent. This activity was blocked using an anti-Fc.gamma.RIII antibody, supporting the role of Fc.gamma.RIII binding in this increase. In addition, ***cell*** binding assays showed the modified antibody bound better to Fc.gamma.RIII-expressing ***cells*** . The increase in ADCC activity is therefore likely due to an increased affinity of the modified antibody for the Fc.gamma.RIII receptor. .COPYRGT. 2001 John Wiley & Sons, Inc.

L5 ANSWER 27 OF 206 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2001114976 EMBASE <<LOGINID::20070409>>
TI Transgenic pigs with human N- ***acetylglucosaminyltransferase*** ***III*** .
AU Miyagawa S.; Murakami H.; Murase A.; Nakai R.; Koma M.; Koyota S.; Matsunami K.; Takahagi Y.; Fujimura T.; Shigehisa T.; Nagashima H.; Shirakura R.; Taniguchi N.
CS Dr. S. Miyagawa, Division of Organ Transplantation, Biomedical Research Center, Osaka Univ. Graduate Sch. Medicine, 2-2 Yamadaoka, Suita, Osaka 565-0871, Japan
SO Transplantation Proceedings, (2001) Vol. 33, No. 1-2, pp. 742-743. .
Refs: 6
ISSN: 0041-1345 CODEN: TRPPA8

PUI S 0041-1345(00)02232-6
CY United States
DT Journal; Conference Article
FS 022 Human Genetics
026 Immunology, Serology and Transplantation
029 Clinical Biochemistry
LA English
ED Entered STN: 30 Apr 2001
Last Updated on STN: 30 Apr 2001
DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

L5 ANSWER 28 OF 206 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2001094002 EMBASE <<LOGINID::20070409>>
TI A new .beta.-1,2-N-acetylglucosaminyltransferase that may play a role in the biosynthesis of mammalian O-mannosyl glycans.
AU Takahashi S.; Sasaki T.; Manya H.; Chiba Y.; Yoshida A.; Mizuno M.; Ishida H.-K.; Ito F.; Inazu T.; Kotani N.; Takasaki S.; Takeuchi M.; Endo T.
CS T. Endo, Department of Glycobiology, Tokyo Metropolitan Inst. Gerontology, 35-2 Sakaecho, Itabashi-ku, Tokyo 173-0015, Japan
SO Glycobiology, (2001) Vol. 11, No. 1, pp. 37-45. .
Refs: 41
ISSN: 0959-6658 CODEN: GLYCE3
CY United Kingdom
DT Journal; Article
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 29 Mar 2001
Last Updated on STN: 29 Mar 2001
AB Recent studies have shown that O-mannosyl glycans are present in several mammalian glycoproteins. Although knowledge on the functional roles of these glycans is accumulating, their biosynthetic pathways are poorly understood. Here we report the identification and initial characterization of a novel enzyme capable of forming GlcNAc.beta.1-2Man linkage, namely UDP-N-acetyl-glucosamine: O-linked mannose .beta.-1,2-N-acetylglucosaminyl-transferase in the microsome fraction of newborn rat brains. The enzyme transfers GlcNAc to .beta.-linked mannose residues, and the formed linkage was confirmed to be .beta.1-2 on the basis of diplococcal .beta.-N-acetylhexosaminidase susceptibility and by high-pH anion-exchange chromatography. Its activity is linearly dependent on time, protein concentration, and substrate concentration and is enhanced in the presence of manganese ion. Its activity is not due to UDP-N-acetylglucosamine: .alpha.-3-D-mannoside .beta.-1,2-N-acetylglucosaminyl- transferase I (GnT-I) or UDP-N-acetylglucosamine: .alpha.-6-D-mannoside .beta.-1,2-D- ***acetylglucosaminyltransferase*** ***II*** (GnT- ***II***), which acts on the early steps of N-glycan biosynthesis, because GnT-I or GnT-II expressed in yeast ***cells*** did not show any GlcNAc transfer activity against a synthetic mannosyl peptide. Taken together, the results suggest that the GlcNAc transferase activity described here is relevant to the O-mannosyl glycan pathway in mammals.

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AN 2001022366 EMBASE <<LOGINID::20070409>>
TI The addition of bisecting N-acetylglucosamine residues to E-cadherin down-regulates the tyrosine phosphorylation of .beta.-catenin.
AU Kitada T.; Miyoshi E.; Noda K.; Higashiyama S.; Ihara H.; Matsuura N.; Hayashi N.; Kawata S.; Matsuzawa Y.; Taniguchi N.
CS N. Taniguchi, Department of Biochemistry, Osaka Univ. Graduate Sch. of Med., 2-2 Yamadaoka, Suita, Osaka 565-0871, Japan. proftani@biochem.med.osaka-u.ac.jp
SO Journal of Biological Chemistry, (5 Jan 2001) Vol. 276, No. 1, pp. 475-480. .
Refs: 35
ISSN: 0021-9258 CODEN: JBCHA3
CY United States
DT Journal; Article
FS 016 Cancer
029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 1 Feb 2001
Last Updated on STN: 1 Feb 2001
AB The enzyme GnT-III (.beta.1,4-N- ***acetylglucosaminyltransferase*** ***III***) catalyzes the addition of a bisecting N-acetylglucosamine (GlcNAc) residue on glycoproteins. Our previous study described that the transfection of GnT-III into mouse melanoma ***cells*** results in the enhanced expression of E-cadherin, which in turn leads to the suppression of lung metastasis. It has recently been proposed that the phosphorylation of a tyrosine residue of .beta.-catenin is associated with ***cell*** migration. The present study reports on the importance of bisecting GlcNAc residues by GnT-III on tyrosine phosphorylation of .beta.-catenin using three types of cancer ***cell*** lines. An addition of bisecting GlcNAc residues to E-cadherin leads to an alteration in ***cell*** morphology and the localization of .beta.-catenin after epidermal growth factor stimulation. These changes are the result of a down-regulation in the tyrosine phosphorylation of .beta.-catenin. In addition, tyrosine phosphorylation of .beta.-catenin by transfection of constitutively active c-src was suppressed in GnT-III transfectants as well as in the case of epidermal growth factor stimulation. Treatment with tunicamycin abolished any differences in .beta.-catenin phosphorylation for the mock vis a vis the GnT-III transfectants. Thus, the addition of a specific N-glycan structure, the bisecting GlcNAc to E-cadherin-.beta.-catenin complex, down-regulates the intracellular signaling pathway, suggesting its implication in ***cell*** motility and the suppression of cancer metastasis.

L5 ANSWER 30 OF 206 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2000427506 EMBASE <<LOGINID::20070409>>
TI Molecular cloning of cDNA encoding N- ***acetylglucosaminyltransferase*** ***II*** from Arabidopsis thaliana.
AU Strasser R.; Steinkellner H.; Boren M.; Altmann F.; Mach L.; Glossl J.; Mucha J.
CS H. Steinkellner, Zentrum for Angewandte Genetik, Universitat fur Bodenkultur Wien, Muthgasse 18, 1190 Wien, Austria. steink@mail.boku.ac.at
SO Glycoconjugate Journal, (1999) Vol. 16, No. 12, pp. 787-791. .
Refs: 18
ISSN: 0282-0080 CODEN: GLJOEW
CY Netherlands
DT Journal; Article
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 21 Dec 2000
Last Updated on STN: 21 Dec 2000
AB N- ***acetylglucosaminyltransferase*** ***II*** (GnTII, EC 2.4.1.143) is a Golgi enzyme involved in the biosynthesis of glycoproteinbound N-linked oligosaccharides, cetalysing an essential step in the conversion of oligomannose-type to complex Nglycans. GnTII activity has been detected in both animals and plants. However, while cDNAs encoding the enzyme have already been cloned from several mammalian
sources no GnTII homologue has been cloned from plants so far. Here we report the molecular cloning of an Arabidopsis thaliana GnTII cDNA with striking homology to its animal counterparts. The predicted domain structure of A. thaliana GnTII indicates a type II transmembrane protein topology as it has been established for the mammalian variants of the enzyme. Upon expression of A. thaliana GnTII cDNA in the baculovirus/insect ***cell*** system, a recombinant protein was produced that exhibited GnTII activity.

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AN 2000202253 EMBASE <<LOGINID::20070409>>
TI Comparative study of the N-glycans of human monoclonal immunoglobulins M produced by hybridoma and parental ***cells*** .
AU Fukuta K.; Abe R.; Yokomatsu T.; Kono N.; Nagatomi Y.; Asanagi M.; Shimazaki Y.; Makino T.
CS K. Fukuta, Life Science Laboratory, Mitsui Chemicals, Inc., 1144, Togo, Mobara, Chiba 297-0017, Japan. Kazuhiro.Fukuta@mitsui.chem.co.jp
SO Archives of Biochemistry and Biophysics, (1 Jun 2000) Vol. 378, No. 1, pp. 142-150. .
Refs: 28
ISSN: 0003-9861 CODEN: ABBIA4
CY United States
DT Journal; Article
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 30 Jun 2000
Last Updated on STN: 30 Jun 2000
AB ***Cell*** - ***cell*** hybridization is one method of establishing ***cell*** lines capable of producing an abundance of antibodies. In order to clearly characterize antibodies produced by hybridomas, the influence of ***cell*** - ***cell*** hybridization on the glycosylation of produced antibodies should be studied. In this report, we describe structural changes of the N-glycans in immunoglobulin M (IgM) produced by a hybridoma ***cell*** line termed 3-4, which was established through hybridization of an IgM-producing Epstein-Barr virus transformed human B- ***cell*** line termed No. 12, and a human myeloma ***cell*** line termed P109. We analyzed the structures of sugar chains on the constant region of the .mu.- chain of IgMs produced by parental No. 12 ***cells*** and hybridoma 3-4 ***cells*** . In both parental ***cells*** and hybridoma ***cells*** , the predominant structures at Asn171, Asn332, and N395 were fully galactosylated biantennary complex types, with or without core fucose and/or bisecting GlcNAc. However, the amount of bisecting GlcNAc was markedly decreased in the hybridoma ***cells*** . Therefore, the activity of UDP-N-acetylglucosamine:.beta.-D-mannoside .beta.-1,4-N- ***acetylglucosaminyltransferase*** (GnT- ***III***) responsible for the formation of bisecting GlcNAc was measured in parental ***cells*** and hybridoma ***cells*** . No. 12 ***cells*** showed some GnT-III activity, whereas P109 ***cells*** showed no such activity. The corresponding level of activity observed in hybridoma 3-4 ***cells*** was much lower than that in No. 12 ***cells*** . The above results demonstrated a reduction in the intracellular activity of GnT-III in the hybridoma ***cells*** , which was largely due to the influence of P109 ***cells*** . Moreover, the sugar chain structures of IgMs produced by the ***cells*** reflected the level of GnT-III activity. (C) 2000 Academic Press.

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AN 2000156969 EMBASE <<LOGINID::20070409>>
TI Medial Golgi but not late golgi glycosyltransferases exist as high molecular weight complexes. Role of luminal domain in complex formation and localization.
AU Opat A.S.; Houghton F.; Gleeson P.A.
CS P.A. Gleeson, Dept. of Pathology/Immunology, Monash University Medical School, Commercial Rd., Prahran, Vic. 3181, Australia. paul.gleeson@med.monash.edu.au
SO Journal of Biological Chemistry, (21 Apr 2000) Vol. 275, No. 16, pp. 11836-11845. .
Refs: 49
ISSN: 0021-9258 CODEN: JBCHA3
CY United States
DT Journal; Article
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 18 May 2000
Last Updated on STN: 18 May 2000
AB To investigate the organization of Golgi glycosyltransferases and their mechanism of localization, we have compared the properties of a number of medial and late acting Golgi enzymes. The medial Golgi enzymes, N-***acetylglucosaminyltransferase*** I and ***II*** (GnTI and GnTII) required high salt for solubilization and migrated as high molecular weight complexes on sucrose density gradients. In contrast, the late acting Golgi enzymes, .beta.1,4- galactosyltransferase and .beta.1,2-fucosyltransferase, were readily solubilized in low salt and migrated as monomers/dimers by sucrose density gradient centrifugation. Analysis of membrane-bound GnTI chimeras indicates that the formation of high molecular weight complexes does not require the transmembrane domain and cytoplasmic tail sequences of GnTI. Furthermore, a soluble form of GnTI, containing the stem region and catalytic domain, accumulated in the Golgi prior to secretion, in contrast to .beta.1,4- galactosyltransferase. Soluble GnTI, which also associated with high molecular weight complexes, was comparable with membrane-bound GnTI in its ability to glycosylate newly synthesized glycoproteins in vivo. Mutation of charged residues within the stem region of GnTI, known to be important for 'kin recognition', had no effect on the efficiency of Golgi localization, the inclusion into high molecular weight complexes, nor functional activity in vivo. The differences in behavior between the medial and late acting Golgi enzymes may contribute to their differential localization and their ability to glycosylate efficiently in the correct Golgi subcompartment.

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AN 2000152249 EMBASE <<LOGINID::20070409>>
TI Regulation of expression of the human .beta.-1,2-N-***acetylglucosaminyltransferase*** ***II*** gene (MGAT2) by Ets transcription factors.
AU Zhang W.; Revers L.; Pierce M.; Schachter H.
CS H. Schachter, Department of Biochemistry, University of Toronto, Toronto, Ont. M5S 1A8, Canada. harry@sickkids.on.ca
SO Biochemical Journal, (15 Apr 2000) Vol. 347, No. 2, pp. 511-518. .
Refs: 50
ISSN: 0264-6021 CODEN: BIJOAK
CY United Kingdom
DT Journal; Article
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 18 May 2000
Last Updated on STN: 18 May 2000
AB Oncogenic transformation of fibroblasts by the src oncogene has long been known to cause an increase in the size of ***cell*** -surface protein-bound oligosaccharides, owing primarily to increased N-glycan branching mediated by increased .beta.-1,6-N-acetylglucosaminyltransferase V (GnT V) activity. The src-responsive element of the GnT V promoter was localized to Ets-binding sites and the promoter was transcriptionally stimulated by both ets-1 and ets-2 expression. Because GnT V action requires the prior action of .beta.-1,2-N- ***acetylglucosaminyltransfera*** *** se*** ***II*** (GnT ***II***) and the human GnT II promoter contains four putative Ets-binding sites, GnT II might also be under oncogenic control via Ets transcription factors. We now report
<------User Break------>
ndependent of the murine
system. These transfectants also displayed decreased invasiveness into Matrigel and inhibited ***cell*** attachment to collagen and laminin. ***Cell*** growth was not affected. Our results demonstrate a causative role for .beta.1-6 branches in invasion and ***cell*** attachment in the extravasation stage of metastasis.

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AN 95225274 EMBASE <<LOGINID::20070409>>
DN 1995225274
TI Changes of .beta.-1,4-N- ***acetylglucosaminyltransferase*** ***III*** (GnT- ***III***) in patients with leukaemia.
AU Yoshimura M.; Ihara Y.; Taniguchi N.
CS Department of Biochemistry, Osaka University Medical School, 2-2 Yamadaoka,Suita, Osaka 565, Japan
SO Glycoconjugate Journal, (1995) Vol. 12, No. 3, pp. 234-240. .
ISSN: 0282-0080 CODEN: GLJOEW
CY United Kingdom
DT Journal; Article
FS 005 General Pathology and Pathological Anatomy
016 Cancer
029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 22 Aug 1995
Last Updated on STN: 22 Aug 1995
AB Changes in the activity and transcription of UDP-N-acetylglucosamine: .beta.- D-mannoside .beta.-1,4-N- ***acetylglucosaminyltransferase*** ***III*** (GnT- ***III*** : EC 2.4.1.144) were investigated in haematological malignancies. GnT-III activity was elevated in patients with chronic myelogeneous leukaemia in blast crisis (CML-BC) and patients with multiple myeloma (MM); whereas most of the normal healthy subjects and patients with other haematological malignancies, including CML in its chronic phase, showed negligible activity. The GnT-III transcript of leukaemic ***cells*** from various haematological diseases showed a single band with a similar size. The ratio of GnT-III activity per normalized transcript in CML-BC was considerably higher than in the other conditions, which provided the possibility that in CML-BC the transcript or the enzyme protein might be more stable, or that a post-translational modification of the enzyme might enhance its activity. Furthermore, a lectin blot analysis of patient specimens and a lectin fluorescence study of CML ***cell*** lines revealed that E4-PHA binding to surface glycoproteins correlated with GnT-III activity, indicating that more bisecting GlcNAc was added to these glycoproteins, catalysed by elevated GnT-III in CML-BC.

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AN 95225205 EMBASE <<LOGINID::20070409>>
DN 1995225205
TI The human UDP-N-acetylglucosamine: .alpha.-6-D-mannoside-.beta.-1,2-N-***acetylglucosaminyltransferase*** ***II*** gene (MGAT2) - Cloning of genomic DNA, localization to chromosome 14q21, expression in insect ***cells*** and purification of the recombinant protein.
AU Tan J.; D'Agostaro G.A.F.; Bendiak B.; Reck F.; Sarkar M.; Squire J.A.; Leong P.; Schachter H.
CS Department of Biochemistry, Hospital for Sick Children, 555 University Avenue,Toronto, Ont. M5G 1X8, Canada
SO European Journal of Biochemistry, (1995) Vol. 231, No. 2, pp. 317-328. .
ISSN: 0014-2956 CODEN: EJBCAI
CY Germany
DT Journal; Article
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 22 Aug 1995
Last Updated on STN: 22 Aug 1995
AB UDP-GlcNAc:.alpha.-6-D-mannoside [GlcNAc to Man.alpha.1-6] .beta.-1,2-N-***acetylglucosaminyltransferase*** ***II*** (GlcNAc-T ***II*** , EC 2.4.1.143) is a Golgi enzyme catalyzing an essential step in the conversion of oligo-mannose to complex N-glycans. A 12-kb probe from a rat liver cDNA encoding GlcNAc-T II was used to screen a human genomic DNA library in .lambda.EMBL3. Southern analysis of restriction endonuclease digests of positive phage clones identified two hybridizing fragments (3.0 and 3.5 kb) which were subcloned into pBlueScript. The inserts of the resulting plasmids (pHG30 and pHG36) are over-lapping clones containing 5.5 kb of genomic DNA. The pHG30 insert (3.0 kb) contains a 1341-bp open reading frame encoding a 447-amino-acid protein, 250 bp of G+C- rich 5'-upstream sequence and 1.4 kb of 3' downstream sequence. The pHG36 insert (3.5 kb) contains 2.75 kb of 5'-upstream sequence and 750 bp of the 5'-end of the open reading frame. The protein sequence showed the domain structure typical of all previously cloned glycosyltransferases, i.e. a short 9-residue putative cytoplasmic N-terminal domain, a 20-residue hydrophobic non-cleavable putative signal-anchor domain and a 418-residue C-terminal catalytic domain Northern analysis of human tissues showed a major message at 3 kb and minor signals at 2 and 4.5 kb. There is no sequence similarity to any previously cloned glycosyltransferases including human UDP-GlcNAc:.alpha.-3- D-mannoside [GlcNAc to Man.alpha.1-3] .beta.-1,2-N-acetylglucosaminyltransferase I (GlcNAc-T I) which has 445 amino acids with a 418-residue C-terminal catalytic domain. The human GlcNAc-T I and II genes (MGAT1 and MGAT2) map to chromosome bands 5q35 and 14q21, respectively, by fluorescence in situ hybridization. The entire coding regions of human GlcNAc-T I and II are each on a single exon. There is 92% identity between the amino acid sequences of the catalytic domains of human and rat GlcNAc-T II. Southern analysis of restriction enzyme digests of human genomic DNA indicates that there is only a single copy of the MGAT2 gene. The full-length coding region of GlcNAc-T II has been expressed in the baculovirus/Sf9 insect ***cell*** system, the recombinant enzyme has been purified to near homogeneity with a specific activity of about 20 .mu.mol .cntdot. min-1 .cntdot. mg-1 and the product synthesized by the recombinant enzyme has been identified by high-resolution 1H-NMR spectroscopy and mass spectrometry.

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AN 95190778 EMBASE <<LOGINID::20070409>>
DN 1995190778
TI Molecular cloning and expression of cDNA encoding the rat UDP-N-acetylglucosamine:.alpha.-6-D-mannoside .beta.-1,2-N-***acetylglucosaminyltransferase*** ***II*** .
AU D'Agostaro G.A.F.; Zingoni A.; Moritz R.L.; Simpson R.J.; Schachter H.;

Bendiak B.
CS Biomembrane Institute, 201 Elliott Ave. West,Seattle, WA 98119, United States
SO Journal of Biological Chemistry, (1995) Vol. 270, No. 25, pp. 15211-15221.
ISSN: 0021-9258 CODEN: JBCHA3
CY United States
DT Journal; Article
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 18 Jul 1995
Last Updated on STN: 18 Jul 1995
AB UDP-N-acetyl-D-glucosamine:.alpha.-6-D-mannoside .beta.-1,2-N-***acetylglucosaminyltransferase*** ***II*** (EC 2.4.1.143) (GnT II) is a Golgi resident enzyme that catalyzes an essential step in the biosynthetic pathway leading from high mannose to complex N-linked oligosaccharides. Sodium dodecyl sulfate-polyacrylamide gel electrophoresis analysis of the enzyme purified from rat liver revealed a polypeptide of 42 kDa. Amino acid sequences were obtained from the N terminus and a tryptic peptide. Overlapping cDNA clones coding for the full-length rat GnT II were obtained. The complete nucleotide sequence revealed a 1326-base pair open reading frame that codes for a polypeptide of 442 amino acids, including a presumptive N-terminal membrane- anchoring domain. The region of cDNA coding for the C-terminal 389 amino acids of rat GnT II was linked in frame to a cDNA segment encoding the cleavable signal sequence of the human interleukin-2 receptor and transiently expressed in COS-7 ***cells*** . A 77-fold enhancement of GnT II activity over a control carrying the GnT II cDNA out-of-frame was detected in the culture medium at 72 h after transfection. 1H-NMR spectroscopy confirmed that the oligosaccharide synthesized in vitro by the recombinant enzyme was the product of GnT II activity. These data verify the identity of the cloned GnT II cDNA and demonstrate that the C-terminal region of the protein includes the catalytic domain.

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AN 95188663 EMBASE <<LOGINID::20070409>>
DN 1995188663
TI Carbohydrate-deficient glycoprotein syndrome type II - An autosomal recessive N- ***acetylglucosaminyltransferase*** ***II*** deficiency different from typical hereditary erythroblastic multinuclearity, with a positive acidified- serum lysis test (HEMPAS).
AU Charuk J.H.M.; Tan J.; Bernardini M.; Haddad S.; Reithmeier R.A.F.; Jaeken J.; Schachter H.
CS Department of Biochemistry, Research Institute, Hospital for Sick Children, 555 University Avenue,Toronto, Ont. M5G 1X8, Canada
SO European Journal of Biochemistry, (1995) Vol. 230, No. 2, pp. 797-805. .
ISSN: 0014-2956 CODEN: EJBCAI
CY Germany
DT Journal; Article
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 12 Jul 1995
Last Updated on STN: 12 Jul 1995
AB Carbohydrate-deficient glycoprotein syndromes (CDGS) are a family of multisystemic congenital diseases resulting in underglycosylated glycoproteins, suggesting defective N-glycan assembly. Fibroblast extracts from two patients with a recently described variant of this disease (CDGS type II) have previously been shown to have over 98% reduced activity of UDP- GlcNAc:.alpha.-6-D-mannoside .beta.-1,2-N-***acetylglucosaminyltransferase*** ***II*** [GlcNAc- TII; Jaeken, J., Schachter, H., Carchon, H., De Cock, P., Coddeville, B. and Spik, G. (1994) Arch. Dis. Childhood 71, 123-127]. We show in this paper that mononuclear ***cell*** extracts from one of these CDGS type-II patients have no detectable GlcNAc-TII activity and that similar extracts from 12 blood relatives of the patient, including his father, mother and brother, have GlcNAc-TII levels 32-67% that of normal levels (average 50.1%.+-.10.7% SD), consistent with an autosomal recessive disease. The poly(N-acetyllactosamine) content of erythrocyte membrane glycoproteins bands 3 and 4.5 of this CDGS patient were estimated, by tomato lectin blotting, to be reduced by 50% relative to samples obtained from blood relatives and normal controls. Similar to patients with hereditary erythroblastic multinuclearity with a positive acidified-serum lysis test (HEMPAS), erythrocyte membrane glycoproteins in the CDGS patient have increased reactivities with concanavalin A, demonstrating the presence of hybrid or oligomannose carbohydrate structures. However, bands 3 and 4.5 in HEMPAS erythrocytes have almost complete lack of poly(N-acetyllactosamine). Furthermore, CDGS type-II patients have a totally different clinical presentation and their erythrocytes do not show the serology typical of HEMPAS, suggesting that the genetic lesions responsible for these two diseases are possibly different.

L5 ANSWER 73 OF 206 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 95052356 EMBASE <<LOGINID::20070409>>
DN 1995052356
TI High expression of UDP-N-acetylglucosamine: .beta.-D mannoside .beta.-1,4-N- ***acetylglucosaminyltransferase*** ***III*** (GNT-***III***) in chronic myelogenous leukemia in blast crisis.
AU Yoshimura M.; Nishikawa A.; Ihara Y.; Nishiura T.; Nakao H.; Kanayama Y.; Matuzawa Y.; Taniguchi N.
CS Department of Biochemistry, Osaka University Medical School, 2-2 Yamadaoka,Suita City, Osaka 565, Japan
SO International Journal of Cancer, (1995) Vol. 60, No. 4, pp. 443-449. .
ISSN: 0020-7136 CODEN: IJCNAW
CY United States
DT Journal; Article
FS 016 Cancer
025 Hematology
LA English
SL English
ED Entered STN: 14 Mar 1995
Last Updated on STN: 14 Mar 1995
AB The activity and mRNA expression of UDP-N-acetylglucosamine: .beta.-D mannoside .beta.-1,4-N-acetylglucosaminyl transferase III (GnT-III: EC 2.4. 1.144) were investigated in hematological malignancies. GnT-III activity was elevated in patients with chronic myelogenous leukemia (CML) in blast crisis and patients with multiple myeloma (MM), as compared to normal healthy subjects and patients with other hematological malignancies including CML in chronic phase. The GnT-III transcript was the same size in leukemic ***cells*** from various hematological diseases and ***cell*** lines, while expression of the transcript was not found to correlate significantly with enzyme activity, implying that post-translational modification might regulate the activity of GnT-III. Southern-blot analysis showed no significant variation in the structure and position of the GnT-III genome, indicating that the gene is present as a single copy without isoforms. Furthermore, analyses by immunoprecipitation and Western blot revealed that high GnT-III activity in KU812 ***cell*** , a CML ***cell*** line, resulted in an increase in E4-PHA binding to CD45, a major surface glycoprotein of the leukocyte, indicating that more bisecting GlcNAc was added to CD45 catalyzed by elevated GnT-III.

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AN 94282812 EMBASE <<LOGINID::20070409>>
DN 1994282812
TI Synthesis of a di-O-methylated pentasaccharide for use in the assay of N-***acetylglucosaminyltransferase*** ***III*** activity.
AU Khan S.H.; Compston C.A.; Palcic M.M.; Hindsgaul O.
CS Department of Chemistry, University of Alberta,Edmonton, Alta. T6G 2G2, Canada
SO Carbohydrate Research, (1994) Vol. 262, No. 2, pp. 283-295. .
ISSN: 0008-6215 CODEN: CRBRAT
CY Netherlands
DT Journal; Article
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 6 Oct 1994
Last Updated on STN: 6 Oct 1994
AB The biantennary oligosaccharide analogue .beta.-D-GlcpNAc-(1 .fwdarw. 2)-.alpha.-D-Manp-(1 .fwdarw. 3)[.beta.-D-GlcpNAc-(1 .fwdarw. 2)-.alpha.-D-Manp-(1 .fwdarw. 6)]-.beta.-D-Manp-O(CH2)8COOMe (3) is a potential substrate for N-acetylglucosaminyltransferases (GlcNAcTs) III-V which are present in mammalian ***cells*** . The di-O-methylated analogue of 3, .beta.-D-GlcpNAc-(1 .fwdarw. 2)-[4-O-methyl-.alpha.-D-Manp]-(1 .fwdarw. 3)-[.beta.-D-GlcpNAc-(1 .fwdarw. 2)-[6-O-methyl-.alpha.-D-Manp]-(1 .fwdarw. 6)]-.beta.-D-Manp-O(CH2)8COOMe (5), was prepared by a block synthesis approach involving sequential addition of two O-methylated disaccharide donors to a protected central .beta.-D-Man residue. The OH groups acted on by GlcNAcT-IV and -V are protected from glycosylation in 5 since they are present as their methyl ethers. Pentasaccharide 5 was found to be an excellent substrate for GlcNAcT-III (EC 2.4.1.144) from rat kidney with K(m) = 0.15 mM. The product formed by incubation of 5 with a rat kidney extract, in the presence of UDP-GlcNAc, was isolated, structurally characterized by NMR spectroscopy and confirmed to be the expected di-O-methyl hexasaccharide where a .beta.-D-GlcpNAc residue had been added to OH-4 of the central .beta.-D-Manp unit.

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AN 94159450 EMBASE <<LOGINID::20070409>>
DN 1994159450
TI Synthesis of tetrasaccharide analogues of the N-glycan substrate of .beta.-(1 .fwdarw. 2)-N- ***acetylglucosaminyltransferase*** ***II*** using trisaccharide precursors and recombinant .beta.-(1 .fwdarw. 2)-N-acetylglucosaminyltransferase I.
AU Reck F.; Springer M.; Paulsen H.; Brockhausen I.; Sarkar M.; Schachter H.
CS Research Institute, Hospital for Sick Children,Toronto, Ont. M5G 1X8, Canada
SO Carbohydrate Research, (1994) Vol. 259, No. 1, pp. 93-101. .
ISSN: 0008-6215 CODEN: CRBRAT
CY Netherlands
DT Journal; Article
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 22 Jun 1994
Last Updated on STN: 22 Jun 1994
AB Recombinant rabbit UDP-GlcNAc : .alpha.-Man-(1 .fwdarw. 3R) .beta.-(1 .fwdarw. 2)-N-acetylglucosaminyltransferase I (EC 2.4.1.101, GlcNAc-T I) produced in the Sf9 insect ***cell*** /baculovirus expression system has been used to convert compounds of the form 3-R-.alpha.-Man(1 .fwdarw.

6)(.alpha.-Man(1 .fwdarw. 3)).beta.-Man-O-octyl to 3-R-.alpha.-Man(1 .fwdarw. 6)(.beta.-GlcNAc(1 .fwdarw. 2).alpha.-Man(1 .fwdarw. 3)).beta.-Man-O-octyl where R is OH (14), O-methyl (17), O-pentyl (18), O-(4,4-azo)pentyl (19), O-(5-iodoacetamido)pentyl (20) and O-(5-amino)pentyl (21); 2-deoxy-.alpha.-Man(1 .fwdarw. 6)(.beta.-GlcNAc(1 .fwdarw. 2).alpha.-Man(1 .fwdarw. 3)).beta.-Man-O-octyl (16), 4-O-methyl-.alpha.-Man(1 .fwdarw. 6)(.beta.-GlcNAc(1 .fwdarw. 2).alpha.-Man(1 .fwdarw. 3)).beta.-Man-O-octyl (22), 6-O-methyl-.alpha.-Man(1 .fwdarw. 6)(.beta.-GlcNAc(1 .fwdarw. 2).alpha. Man(1 .fwdarw. 3)).beta.-Man-O-octyl (23) and .alpha.-Man(1 .fwdarw. 6)[.beta.-GlcNAc(1 .fwdarw. 2)(4-O-methyl).alpha.-Man(1 .fwdarw. 3)].beta.-Man-O-octyl (15) were also synthesized by this procedure. The yields ranged from 80 to 99%. Products were characterized by high resolution 1H and 13C nuclear magnetic resonance spectroscopy and fast atom bombardment mass spectrometry. Compounds 14, 15, 17, 22, and 23 are excellent substrates for UDP-GlcNAc: .alpha.-Man(1 .fwdarw. 6R) .beta.-(1 .fwdarw. 2)-N- ***acetylglucosaminyltransferase*** ***II*** and the other compounds are inhibitors of this enzyme.

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AN 94125629 EMBASE <<LOGINID::20070409>>
DN 1994125629
TI Selective suppression of N- ***acetylglucosaminyltransferase*** ***III*** activity in a human hepatoblastoma ***cell*** line transfected with hepatitis B virus.
AU Miyoshi E.; Nishikawa A.; Ihara Y.; Hayashi N.; Fusamoto H.; Kamada T.; Taniguchi N.
CS Department of Biochemistry, Osaka University Medical School, 2-2 Yamadaoka,Suita, Osaka 565, Japan
SO Cancer Research, (1994) Vol. 54, No. 7, pp. 1854-1858. .
ISSN: 0008-5472 CODEN: CNREA8
CY United States
DT Journal; Article
FS 016 Cancer
029 Clinical Biochemistry
048 Gastroenterology
LA English
SL English
ED Entered STN: 11 May 1994
Last Updated on STN: 11 May 1994
AB UDP-N-acetylglucosamine:.beta.-D-mannoside .beta.-1,4-N- ***acetylglucosaminyltransferase*** ***III*** (GnT- ***III***) is a key enzyme in the branching of asparagine-linked oligosaccharides, which are present in surface membrane proteins of various tissues and in secretory glycoproteins. The activity of GnT-III was assayed in 2 human hepatoblastoma ***cell*** lines, Huh6, which was the parental ***cell*** line, and HB611, which was established by transfection of 3 tandem copies of the hepatitis B virus genome into Huh6. A significant difference in GnT-III activity was found between Huh6 and HB611 (136 .+-. 18.3 pmol/h/mg versus 6.7 .+-. 2.4 pmol/h/mg; mean .+-. SD, P < 0.001), whereas levels of the glycosyltransferases .alpha.-3-D-mannoside .beta.-1,4-N- acetylglucosaminyltransferase IV, .alpha.-6-D-mannoside .beta.-1,6-N- acetylglucosaminyltransferase-V, and .beta.-1,4-galactosyltransferase were almost the same in both ***cell*** lines. Northern blot analysis indicated that the decreased activity of GnT-III in HB611 was due to the decreased transcript. When HB611 was treated with interferon-.alpha., expression of hepatitis B virus- related mRNA decreased, and the activity of GnT-III increased from 8.5 .+-. 3.8 to 22.0 .+-. 7.2 pmol/h/mg (mean .+-. SD, P < 0.05). This increase was not found in Huh6. Binding capacity with erythrocyte phytohemagglutinin in these ***cells*** using fluorescence-activated ***cell*** sorter analysis was different, suggesting that the structure of sugar chain on the ***cell*** surface might be altered by suppression of GnT-III activity. This is the first report that hepatitis B virus selectively suppressed the GnT-III activity in hepatoblastoma ***cells*** .

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AN 94066403 EMBASE <<LOGINID::20070409>>
DN 1994066403
TI Isolation of a matrix that binds medial Golgi enzymes.
AU Slusarewicz P.; Nilsson T.; Hui N.; Watson R.; Warren G.
CS Cell Biology Laboratory, Imperial Cancer Research Fund,London WC2A 3PX, United Kingdom
SO Journal of Cell Biology, (1994) Vol. 124, No. 4, pp. 405-413. .
ISSN: 0021-9525 CODEN: JCLBA3
CY United States
DT Journal; Article
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 25 Mar 1994
Last Updated on STN: 25 Mar 1994
AB Rat liver Golgi stacks were extracted with Triton X-100 at neutral pH. After centrifugation the low speed pellet contained two medial-Golgi enzymes, N- ***acetylglucosaminyltransferase*** I and mannosidase ***II*** , but no enzymes or markers from other parts of the Golgi apparatus. Both were present in the same structures which appeared, by electron microscopy, to be small remnants of cisternal membranes. The enzymes could be removed by treatment with low salt, leaving behind a salt pellet, which we term the matrix. Removal of salt caused specific re-binding of both enzymes to the matrix, with an apparent dissociation constant of 3 nM for mannosidase II. Re-binding was abolished by pretreatment of intact Golgi stacks with proteinase K, suggesting that the matrix was present between the cisternae.

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AN 94027189 EMBASE <<LOGINID::20070409>>
DN 1994027189
TI Processing of asparagine-linked oligosaccharides in insect ***cells*** . N- ***acetylglucosaminyltransferase*** I and ***II*** activities in cultured lepidopteran ***cells*** .
AU Altmann F.; Kornfeld G.; Dalik T.; Staudacher E.; Glossl J.
CS Institut fur Chemie, Universitat fur Bodenkultur, Gregor-Mendelstrasse 33,A-1180 Wien, Austria
SO Glycobiology, (1993) Vol. 3, No. 6, pp. 619-625. .
ISSN: 0959-6658 CODEN: GLYCE3
CY United Kingdom
DT Journal; Article
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 20 Feb 1994
Last Updated on STN: 20 Feb 1994
AB The levels of .beta.1,2-N-acetylglucosaminyltransferase (GlcNAc-T) I and II activities in cultured ***cells*** from Bombyx mori (Bm-N), Mamestra brassicae (IZD-Mb-0503) and Spodoptera frugiperda (Sf-9 and Sf-21) were investigated. Apart from initial experiments with Man.alpha.-3(Man.alpha.1-6)Man.beta.1-O(CH2)8COOH3 and 3H-labelled UDP-GlcNAc as substrates, GlcNAc-T I activity was measured with a nonradioactive HPLC method using pyridylaminated Man3GlcNAc2 and Man5GlcNAc2 as acceptor oligosaccharides. It was shown by reversed-phase HPLC, exoglycosidase digestion and methylation analysis that the product obtained with Man3GlcNAc2 contained a terminal GlcNAc residue linked .beta.1,2 to the .alpha.1,3 arm of the acceptor. Compared to the enzyme from the human hepatoma ***cell*** line HepG2, insect ***cell*** GlcNAc-T I exhibited a much higher preference for the Man5 substrate. The GlcNAc-T I from Mb-0503 ***cells*** had apparent K(m) and V(max) values for pyridylaminated Man3- and Man5GlcNAc2 of 2.15 and 0.21 mM, and of 3.4 and 11.4 nmol/h/mg of ***cell*** protein, respectively. When Man5GlcNAc2 was used as the acceptor substrate, the levels of GlcNAc-T I activity in the four insect ***cell*** lines ranged between 7.5 and 14.7 nmol/h/mg of ***cell*** protein, and thus were comparable to that of HepG2 ***cells*** . Evidence is presented for the dependence of lepidopteran fucosyltransferase on the presence of terminal N-acetylglucosamine. GlcNAc-T II activity could be demonstrated by HPLC using GlcNAc.beta.1-2Man.alpha.1-3(Man.alpha.1-6)Man.beta.1-4GlcNAc.beta.1-4GlcNAc-pyridylamine as the acceptor in the presence of 6-acetamido-6-deoxycastanospermine as an inhibitor of .beta.-N-acetylglucosaminidase. However, the insect ***cells*** exhibited specific activities of GlcNAc-T II of only 0.02-0.11 nmol/h/mg of ***cell*** protein, much less than HepG2 ***cells*** .

L5 ANSWER 79 OF 206 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 92285236 EMBASE <<LOGINID::20070409>>
DN 1992285236
TI Purification, cDNA cloning, and expression of UDP-N-acetylglucosamine:.beta.- D-mannoside .beta.-1,4N- ***acetylglucosaminyltransferase*** ***III*** from rat kidney.
AU Nishikawa A.; Ihara Y.; Hatakeyama M.; Kangawa K.; Taniguchi N.
CS Department of Biochemistry, Osaka University Medical School, 2-2 Yamadaoka,Suita, Osaka 565, Japan
SO Journal of Biological Chemistry, (1992) Vol. 267, No. 25, pp. 18199-18204. .
ISSN: 0021-9258 CODEN: JBCHA3
CY United States
DT Journal; Article
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 11 Oct 1992
Last Updated on STN: 11 Oct 1992
AB UDP-N-acetylglucosamine:.beta.-D-mannoside .beta.-1,4N- ***acetylglucosaminyltransferase*** ***III*** (GnT- ***III*** : EC 2.4.1.144) catalyzes the addition of N-acetylglucosamine in .beta.1-4 linkage to the .beta.-linked mannose of the trimannosyl core of N-linked sugar chains. The enzyme has been purified over 153,000-fold in 1.5% yield from a Triton X-100 extract of rat kidney by fractionation procedures utilizing QAE-Sepharose, Cu2+-chelating Sepharose, and affinity chromatography on UDP-hexanolamine and substrate-conjugated Sepharose. The purified protein migrates as one major and one minor band with apparent molecular masses of 62 kDa and 52 kDa, respectively. The purified enzyme was digested with trypsin, and the amino acid sequences of four peptides were determined. Oligonucleotide primers were designed according to those amino acid sequences and used in the polymerase chain reaction. Screening for the cDNA for GnT-III was carried out by plaque hybridization using a rat kidney cDNA library (.lambda.gt10) and a polymerase chain reaction product as the probe. Rat kidney GnT-III has 536 amino acids and three putative N-glycosylation sites. There is no sequence homology to other previously cloned glycosyltransferases, but the enzyme appears to be a type II transmembrane protein like the other glycosyltransferases. The GnT-III activity in transiently transfected

COS-1 ***cells*** was found to be about 500- 3600-fold as compared to that in non- or mock-transfected ***cells*** .

L5 ANSWER 80 OF 206 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 92162610 EMBASE <<LOGINID::20070409>>
DN 1992162610
TI Enzymatic basis of sugar structures of .alpha.-fetoprotein in hepatoma and hepatoblastoma ***cell*** lines: Correlation with activities of .alpha.1-6 fucosyltransferase and N-acetylglucosaminyltransferases III and V.
AU Ohno M.; Nishikawa A.; Koketsu M.; Taga H.; Endo Y.; Hada T.; Higashino K.; Taniguchi N.
CS Department of Biochemistry, Osaka University Medical School, 2-2 Yamadaoka,Osaka 565, Japan
SO International Journal of Cancer, (1992) Vol. 51, No. 2, pp. 315-317. . ISSN: 0020-7136 CODEN: IJCNAW
CY United States
DT Journal; Article
FS 016 Cancer
029 Clinical Biochemistry
048 Gastroenterology
LA English
SL English
ED Entered STN: 28 Jun 1992
Last Updated on STN: 28 Jun 1992
AB .alpha.-Fetoproteins (AFPs) were purified from 2 hepatoma ***cell*** lines (Hep G2 and HuH-7) and a hepatoblastoma ***cell*** line (HuH-6), and the structures of pyridylaminated (PA) derivatives of their sugar chains were analyzed by HPLC. Simultaneously, the activities of .alpha.1-6 fucosyltransferase (.alpha.1-6FT) and N-***acetylglucosaminyltransferase*** ***III*** (GnT- ***III***), IV (GnT-IV) and V (GnT-V) were assayed in these ***cell*** lines. For all 3 ***cell*** lines the major sugar chain detected was a fucosylated biantennary structure. Hep G2 ***cells*** contained a high level of GnT-V, which catalyzes the formation of a tri'-antennary structure, and in fact a substantial percentage of the AFP sugar chains in these ***cells*** had the tri'-antennary structure. .alpha.1-6FT was also high, and fucosylated tri'-structures were detected, which suggests that high activities of transferases affect the AFP sugar chains. In HuH-6 ***cells*** , GnT-III, which catalyzes the formation of bisecting GlcNAc, was elevated. Correspondingly, a fucosylated, bisected biantennary structure was found as a major sugar chain. In the HuH-7 ***cell*** line, the contents of bisecting GlcNAc and tri' structure were low and neither GnT-III nor GnT-V was elevated. These data indicate that the sugar structures of AFP in these ***cell*** lines correlate well with the activities of .alpha.1-6 FT, GnT-III and GnT-V.

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AN 92035967 EMBASE <<LOGINID::20070409>>
DN 1992035967
TI Enzymatic amplification involving glycosyltransferases forms the basis for the increased size of asparagine-linked glycans at the surface of NIH 3T3 ***cells*** expressing the N-ras proto-oncogene.
AU Easton E.W.; Bolscher J.G.M.; Van den Eijnden D.H.
CS Dept of Medical Chemistry, Vrije Universiteit, Van der Boechorststraat 7,NL-1081 BT Amsterdam, Netherlands
SO Journal of Biological Chemistry, (1991) Vol. 266, No. 32, pp. 21674-21680. . ISSN: 0021-9258 CODEN: JBCHA3
CY United States
DT Journal; Article
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 20 Mar 1992
Last Updated on STN: 20 Mar 1992
AB Expression of ras oncogenes in NIH 3T3 fibroblasts results in the acquisition by these ***cells*** of an invasive potential concomitant with the appearance of ***cell*** surface asparagine-linked complex-type glycan structures of a higher average molecular weight (Bolscher, J. G. M., van der Bijl, M. M. W., Neefjes, J. J., Hall, A., Smets, L. A., and Ploegh, H. L. (1988) EMBO J. 7, 3361-3368). We have investigated the enzymatic basis for the altered glycosylation by assessing the activities of all major Golgi glycosyltransferases involved in the synthesis of these structures. Use was made of a stable transfectant ***cell*** line (T15) containing the N-ras-protooncogene under the control of a glucocorticoid-inducible mouse mammary tumor virus promoter. Upon induction of the ras gene with dexamethasone: 1) the levels of N- ***acetylglucosaminyltransferase*** I and ***II*** were essentially unaltered, indicating an unaffected potential to synthesize complex-type glycans; 2) the activities of the branching N-***acetylglucosaminyltransferase*** ***III*** and V were elevated 2- to 2.5-fold suggesting the formation of increased amounts of bisected glycans and of structures carrying a Gal.beta.1.fwdarw.4GlcNAc.beta.1.fwdarw.6Man-branch; 3) the levels of the elongating .beta.4-galactosyltransferase and .beta.3-N-acetylglucosaminyl-transferase were increased 5- to 7-fold indicating a strongly enhanced capacity to synthesize polylactosaminoglycan chains; 4) the level of the major chain-terminating enzyme, .alpha.3-galactosyltransferase, was slightly decreased (0.7-fold), whereas those of the .alpha.3- and .alpha.6-sialytransferases were slightly elevated (1.3- and 2-fold, respectively), suggesting a shift from termination by .alpha.-galactosyl residues to termination by sialic acid moieties. Studies on the acceptor specificities of the different glycosyltransferases indicate that these changes occur in a coordinated manner in which the effects of altered glycosyltransferase expression levels amplify each other. Analysis of the size of ***cell*** surface complex-type glycopeptides before and after digestion with neuraminidase and endo-.beta.-galactosidase suggested an increased sialic acid density, an increase in the number and/or length of polylactosaminoglycan chains, and an increased branching of the glycans upon N-ras induction. The enzymatic results explain these structural changes and allow us to define the alterations in glycosylation pathways associated with ras expression.

L5 ANSWER 82 OF 206 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 91197219 EMBASE <<LOGINID::20070409>>
DN 1991197219
TI A subclass of ***cell*** surface carbohydrates revealed by a CHO mutant with two glycosylation mutations.
AU Stanley P.; Sundaram S.; Sallustio S.
CS Department of Cell Biology, Albert Einstein College of Medicine, 1300 Morris Park Avenue, Bronx, NY 10461, United States
SO Glycobiology, (1991) Vol. 1, No. 3, pp. 307-314. . ISSN: 0959-6658 CODEN: GLYCE3
CY United Kingdom
DT Journal; Article
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 16 Dec 1991
Last Updated on STN: 16 Dec 1991
AB A novel lectin-resistance phenotype was displayed by a LEC10 Chinese hamster ovary (CHO) ***cell*** mutant that was selected for resistance to the erythroagglutinin, E-PHA. Biochemical and genetic analyses revealed that the phenotype results from the expression of two glycosylation mutations, LEC10 and lec8. The LEC10 mutation causes the appearance of A- ***acetylglucosaminyltransferase*** ***III*** (GlcNAc-TIII) activity and the production of N-linked carbohydrates with a bisecting GlcNAc residue. The lec8 mutation inhibits translocation of UDP-Gal into the Golgi lumen and thereby dramatically reduces galactosylation of all glycoconjugates. This reduction in galactose addition does not, however, cause Lec8 mutants to be very resistant to the galactose-binding lectin, ricin. By contrast, the double mutant EEC10.Lec8 behaved like a LEC10 mutant and was highly resistant to ricin. Based on structural studies of ***cellular*** glycopeptides as well as glycopeptides of the G glycoprotein of vesicular stomatitis virus grown in mutant ***cells*** , it appears that the ricin resistance of LEC10.Lec8 ***cells*** is due to the presence of a small number of Gal residues on branched, N-linked carbohydrates that also carry the bisecting GlcNAc residue. Labelling of N-linked ***cellular*** carbohydrates with [3H]galactose was found to occur at a low level for a wide spectrum of ***cellular*** glycoproteins in independent Lec8 mutants. Studies of the LEC10.Lec8 mutant have, therefore, led to the identification of a subset of structures that are acceptors for Gal when intra-Golgi UDP-Gal levels are limiting. This mutant also illustrates the potential for regulating ***cell*** surface recognition by carbohydrate-binding proteins by altering the expression of a single glycosyltransferase such as GlcNAc-TIII.

L5 ANSWER 83 OF 206 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 91044727 EMBASE <<LOGINID::20070409>>
DN 1991044727
TI HEMPAS disease: Genetic defect of glycosylation.
AU Fukuda M.N.
CS La Jolla Cancer Research Foundation, 10901 North Torrey Pines Road, La Jolla, CA 92037, United States
SO Glycobiology, (1990) Vol. 1, No. 1, pp. 9-15. . ISSN: 0959-6658 CODEN: GLYCE3
CY United Kingdom
DT Journal; (Short Survey)
FS 022 Human Genetics
025 Hematology
029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 16 Dec 1991
Last Updated on STN: 16 Dec 1991
AB Congenital dyserythropoietic anaemia Type II or HEMPAS (hereditary erythroblastic multinuclearity with positive acidified serum lysis test) is a rare genetic anaemia in humans, inherited in an autosomally recessive mode. Biochemical analyses of HEMPAS erythrocyte membranes suggested strongly that HEMPAS is caused by defective glycosylation of erythrocyte membrane glycoproteins. Most recently a HEMPAS case has been identified as being defective in the gene encoding Golgi .alpha.-mannosidase II by using cDNA probe of .alpha.-mannosidase II. At present, it is not clear whether HEMPAS is a genetically heterogenous collection of glycosylation deficiencies, as some HEMPAS cases showed a low level of N-***acetylglucosaminyltransferase*** ***II*** . Abnormal glycosylation of serum glycoproteins and association of liver cirrhosis in HEMPAS patients indicate that HEMPAS disease is not restricted to erythroid ***cells*** . On the other hand, normal development of HEMPAS patients during embryonic stage strongly suggests the possibilities

of fetal type isozyme in place of defective glycosylation enzyme.

L5 ANSWER 84 OF 206 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 91000191 EMBASE <<LOGINID::20070409>>
DN 1991000191
TI Modulation of N- ***acetylglucosaminyltransferase*** ***III*** , IV and V activities and alteration of the surface oligosaccharide structure of a myeloma ***cell*** line by interleukin 6.
AU Nakao H.; Nishikawa A.; Karasuno T.; Nishiura T.; Iida M.; Kanayama Y.; Yonezawa T.; Tarui S.; Taniguchi N.
CS Second Department of Internal Medicine, Osaka University Medical School, 1-1-50, Fukushima, Fukushimaku, Osaka 553, Japan
SO Biochemical and Biophysical Research Communications, (1990) Vol. 172, No. 3, pp. 1260-1266. .
ISSN: 0006-291X CODEN: BBRCA
CY United States
DT Journal; Article
FS 016 Cancer
029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 16 Dec 1991
Last Updated on STN: 16 Dec 1991
AB The activity of N- ***acetylglucosaminyltransferase*** (GnT) ***III*** , IV and V on a myeloma ***cell*** line, OPM-1, was examined after incubation with interleukin 6 (IL-6). While augmenting ***cell*** proliferation, IL-6 resulted in a decrease of GnT III activity and an increase of GnT IV and V activities. Consistent with this, OPM-1 cultured with IL-6 showed an increased affinity to Datura stramonium lectin, which recognizes asialo-tri- and asialo-tetraantenary N-linked oligosaccharides. These results indicate that IL-6 modulates glycosyltransferase activity and the oligosaccharide structure of target ***cells*** .

L5 ANSWER 85 OF 206 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 90347643 EMBASE <<LOGINID::20070409>>
DN 1990347643
TI Incomplete synthesis of N-glycans in congenital dyserythropoietic anemia type II caused by a defect in the gene encoding .alpha.-mannosidase II.
AU Fukuda M.N.; Masri K.A.; Dell A.; Luzzatto L.; Moremen K.W.
CS La Jolla Cancer Research Foundation, La Jolla, CA 92037, United States
SO Proceedings of the National Academy of Sciences of the United States of America, (1990) Vol. 87, No. 19, pp. 7443-7447. .
ISSN: 0027-8424 CODEN: PNASA6
CY United States
DT Journal; Article
FS 022 Human Genetics
029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 13 Dec 1991
Last Updated on STN: 13 Dec 1991
AB Congenital dyserythropoietic anemia type II, or hereditary erythroblastic multinuclearity with a positive acidified-serum-lysis test (HEMPAS), is a genetic anemia in humans inherited by an autosomally recessive mode. The enzyme defect in most HEMPAS patients has previously been proposed as a lowered activity of N- ***acetylglucosaminyltransferase*** ***II*** , resulting in a lack of polylactosamine on proteins and leading to the accumulation of polylactosaminyl lipids. A recent HEMPAS case, G.C., has now been analyzed by ***cell*** -surface labeling, fast-atom-bombardment mass spectrometry of glycopeptides, and activity assay of glycosylation enzymes. Significantly decreased glycosylation of polylactosaminoglycan proteins and incompletely processed asparagine-linked oligosaccharides were detected in the erythrocyte membranes of G.C. In contrast to the earlier studied HEMPAS cases, G.C. ***cells*** are normal in N- ***acetylglucosaminyltransferase*** ***II*** activity but are low in .alpha.-mannosidase II (.alpha.-ManII) activity. Northern (RNA) analysis of poly(A)+ mRNA from normal, G.C., and other unrelated HEMPAS ***cells*** all showed double bands at the 7.6-kilobase position, detected by an .alpha.-ManII cDNA probe, but expression of these bands in G.C. ***cells*** was substantially reduced (<10% of normal). In Southern analysis of G.C. and normal genomic DNA, the restriction fragment patterns detected by the .alpha.-ManII cDNA probe were indistinguishable. These results suggest that G.C. ***cells*** contain a mutation in .alpha.-ManII-encoding gene that results in inefficient expression of .alpha.-ManII mRNA, either through reduced transcription or message instability. This report demonstrates that HEMPAS is caused by a defective gene encoding an enzyme necessary for the synthesis of asparagine-linked oligosaccharides.

L5 ANSWER 86 OF 206 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 90308897 EMBASE <<LOGINID::20070409>>
DN 1990308897
TI Carbohydrate analysis of immunoglobulin G myeloma proteins by lectin and high performance liquid chromatography: Role of glycosyltransferases in the structures.
AU Nishiura T.; Fujii S.; Kanayama Y.; Nishikawa A.; Tomiyama Y.; Iida M.; Karasuno T.; Nakao H.; Yonezawa T.; Taniguchi N.; Tarui S.
CS 2nd Dept. of Internal Medicine, Osaka Univ. Medical School, 1-1-50 Fukushima,Fukushima-ku, Osaka 553, Japan
SO Cancer Research, (1990) Vol. 50, No. 17, pp. 5345-5350. .
ISSN: 0008-5472 CODEN: CNREA8
CY United States
DT Journal; Article
FS 016 Cancer
025 Hematology
026 Immunology, Serology and Transplantation
029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 13 Dec 1991
Last Updated on STN: 13 Dec 1991
AB The carbohydrate structures and the enzymatic basis for glycosylation of IgG by bone marrow plasma ***cells*** were determined in 7 patients with monoclonal gammopathy of undetermined significance and 22 patients with IgG MM. Lectin-binding analysis showed that in all cases of monoclonal gammopathy of undetermined significance and normal controls the IgG heavy chains bound to Ricinus communis agglutinin more strongly than to concanavalin A. In contrast, the IgG in 11 of the 17 advanced cases of MM (stages II and III) studied reacted to concanavalin A more strongly. Structural analysis showed that the reduced R. communis agglutinin binding capacity of these MM IgGs was due to hypogalactosylation of IgG. The galactosyltransferase and N- ***acetylglucosaminyltransferase*** ***III*** of the bone marrow myeloma ***cells*** from 5 MM cases were found to have a low enzyme activity ratio of galactosyltransferase to N- ***acetylglucosaminyltransferase*** ***III*** which reflects the hypogalactosylation. This indicates that the difference in the carbohydrate moieties observed in myeloma proteins is due to variations in the activities of the two glycosyltransferases.

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AN 90283044 EMBASE <<LOGINID::20070409>>
DN 1990283044
TI Determination of N-acetylglucosaminyltransferases III, IV and V in normal and hepatoma tissues of rats.
AU Nishikawa A.; Gu J.; Fujii S.; Taniguchi N.
CS Department of Biochemistry, Osaka University Medical Sch., 4-3-57 Nakanoshima,Kitaku, Osaka 530, Japan
SO Biochimica et Biophysica Acta - General Subjects, (1990) Vol. 1035, No. 3, pp. 313-318. .
ISSN: 0304-4165 CODEN: BBGSB3
CY Netherlands
DT Journal; Article
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 13 Dec 1991
Last Updated on STN: 13 Dec 1991
AB N- ***Acetylglucosaminyltransferase*** ***III*** , IV and V activities were assayed in various rat tissues and hepatomas using the same fluorescence-labeled sugar chain, GlcNAc.beta.1-2Man.alpha.1-3-(GlcNAc.beta.1-2Man.alpha.1-6)Man.beta.1-4GlcNAc.beta.1-4GlcNAc-2-aminopyridine as a substrate. The N- ***acetylglucosaminyltransferase*** ***III*** activity toward the substrate is the highest in most rat tissues including primary rat hepatoma. A relatively higher activity for GnT-V is found in small intestine, serum and hepatoma as compared to that of GnT-IV. Some kinetic properties of these enzymes in crude extracts were also determined.

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AN 90148477 EMBASE <<LOGINID::20070409>>
DN 1990148477
TI Structural heterogeneity of sugar chains in immunoglobulin G. Conformation of immunoglobulin G molecule and substrate specificities of glycosyltransferases.
AU Fujii S.; Nishiura T.; Nishikawa A.; Miura R.; Taniguchi N.
CS Department of Biochemistry, Osaka University, Medical School,Osaka 530, Japan
SO Journal of Biological Chemistry, (1990) Vol. 265, No. 11, pp. 6009-6018. .
ISSN: 0021-9258 CODEN: JBCHA3
CY United States
DT Journal; Article
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 13 Dec 1991
Last Updated on STN: 13 Dec 1991
AB The heterogeneous asparagine-linked sugar chains of bovine and human immunoglobulins G were separated into 12 components by reversed-phase high performance liquid chromatography, and their structures were determined by 1H NMR spectroscopy. Both immunoglobulin (Ig) G sources contained eight non-bisected biantennary complexes and four bisected biantennary complexes. In the non-bisected sugar chains of bovine IgG, galactosylation of the Man.alpha.1-3 branch predominated over that of the Man.alpha.1-6, whereas in the bisected complexes galactosylation of the Man.alpha.1-6 branch predominated. This difference can be explained by the substrate specificities of the galactosyltransferases and of the N- ***acetylglucosaminyltransferase*** ***III*** involved in their synthesis. The sugar chains of human IgG1 differs in the distribution of its galactose residues from bovine IgG and human IgG2. The Man.alpha.1-6 branch of all IgG1s was more highly galactosylated than the Man.alpha.1-3

branch even in the non-bisected complexes. Such findings are in conflict with the substrate specificities of galactosyltransferases. Whereas these enzymes derivatized more of the Man.alpha.1-6 branch of native human IgG1, in denatured protein more of the Man.alpha.1-3 branch was galactosylated. Thus, protein conformation may influence the structure of its sugar chains.

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AN 90009403 EMBASE <<LOGINID::20070409>>
DN 1990009403
TI N- ***Acetylglucosaminyltransferase*** ***III*** in human serum, and liver and hepatoma tissues: Increased activity in liver cirrhosis and hepatoma patients.
AU Ishibashi K.; Nishikawa A.; Hayashi N.; Kasahara A.; Sato N.; Fujii S.; Kamada T.; Taniguchi N.
CS First Department of Medicine, Osaka University Medical School, Fukushima-ku, Osaka, Japan
SO Clinica Chimica Acta, (1989) Vol. 185, No. 3, pp. 325-332. .
ISSN: 0009-8981 CODEN: CCATAR
CY Netherlands
DT Journal; Conference Article
FS 016 Cancer
029 Clinical Biochemistry
048 Gastroenterology
LA English
SL English
ED Entered STN: 13 Dec 1991
Last Updated on STN: 13 Dec 1991
AB An N- ***acetylglucosaminyltransferase*** ***III*** which catalyzes the addition of N-acetylglucosamine through a .beta.1-4 linkage (bisecting N-acetylglucosamine) to the .beta.-linked mannose of the trimannosyl core structure of N-linked oligosaccharides of glycoproteins was measured in human serum, and liver and hepatoma tissues. The enzyme activity in serum was significantly elevated in patients with hepatomas and liver cirrhosis, and the activity markedly decreased on the transcatheter arterial embolization treatment. High activities were also found in the hepatoma and cirrhotic liver tissues, indicating that the serum activity reflected the activity in the tissue. The assaying of the enzyme activity in serum appears to be useful for the detection and monitoring of primary hepatomas.

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AN 89146971 EMBASE <<LOGINID::20070409>>
DN 1989146971
TI N- ***Acetylglucosaminyltransferase*** ***III*** , IV and V activities in Novikoff ascites tumour ***cells*** , mouse lymphoma ***cells*** and hen oviduct. Application of a sensitive and specific assay by use of high-performance liquid chromatography.
AU Koenderman A.H.L.; Koppen P.L.; Koeleman C.A.M.; Van Den Eijnden D.H.
CS Department of Medical Chemistry, Vrije Universiteit, 1007 MC Amsterdam, Netherlands
SO European Journal of Biochemistry, (1989) Vol. 181, No. 3, pp. 651-655. .
ISSN: 0014-2956 CODEN: EJBCAI
CY Germany
DT Journal
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 12 Dec 1991
Last Updated on STN: 12 Dec 1991
AB A specific and fast method for the determination of N- ***acetylglucosaminyltransferase*** ***III*** , IV and V activity in one assay is described. The method is based on the separation by HPLC of the three transferase products formed from the common acceptor oligosaccharide substrate GlcNAc.beta.1 .fwdarw. 2Man.alpha.1 .fwdarw. 3(GlcNAc.beta.1 .fwdarw. 2Man.alpha.1 .fwdarw. 6)Man.beta.1 .fwdarw. 4GlcNAc. Assays are not interfered with by substances that result from enzymatic or chemical breakdown of the donor substrate UDP-[14C]GlcNAc. Using this assay system N- ***acetylglucosaminyltransferase*** ***III*** , IV and V activities were estimated in Novikoff ascites tumour ***cells*** , mouse lymphoma BW 5147 ***cells*** and hen oviduct.

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AN 89123487 EMBASE <<LOGINID::20070409>>
DN 1989123487
TI Expression of N- ***acetylglucosaminyltransferase*** ***III*** in hepatic nodules generated by different models of rat liver carcinogenesis.
AU Pascale R.; Narasimhan S.; Rajalakshmi S.
CS Department of Pathology, Medical Sciences Building, University of Toronto, Toronto, Ont. M5S 1A8, Canada
SO Carcinogenesis, (1989) Vol. 10, No. 5, pp. 961-964. .
ISSN: 0143-3334 CODEN: CRNGDP
CY United Kingdom
DT Journal
FS 016 Cancer
029 Clinical Biochemistry
048 Gastroenterology
LA English
ED Entered STN: 12 Dec 1991
Last Updated on STN: 12 Dec 1991

DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

L5 ANSWER 92 OF 206 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 89107481 EMBASE <<LOGINID::20070409>>
DN 1989107481
TI Defective glycosylation of erythrocyte membrane glycoconjugates in a variant of congenital dyserythropoietic anemia type II: Association of low level of membrane-bound form of galactosyltransferase.
AU Fukuda M.N.; Masri K.A.; Dell A.; Thonar E.J.-M.; Klier G.; Lowenthal R.M.
CS La Jolla Cancer Research Foundation, La Jolla, CA 92037, United States
SO Blood, (1989) Vol. 73, No. 5, pp. 1331-1339. .
ISSN: 0006-4971 CODEN: BLOOAW
CY United States
DT Journal
FS 007 Pediatrics and Pediatric Surgery
022 Human Genetics
025 Hematology
029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 12 Dec 1991
Last Updated on STN: 12 Dec 1991
AB Congenital dyserythropoietic anemia type II (CDA II) or HEMPAS is a genetic disease caused by plasma membrane abnormality. The enzymic defect of HEMPAS has been suggested to be the lowered activity of N- ***acetylglucosaminyltransferase*** ***II*** , resulting in lack of polylactosamine formation on proteins and leading to accumulation of polylactosaminyl lipids. In contrast to typical HEMPAS cases, ***cell*** -surface labeling of the erythrocytes of a HEMPAS variant G.K. showed an absence of polylactosamines either on proteins or on lipids. Fast-atom bombardment mass spectrometry analysis of G.K.'s erythrocyte glycopeptides detected a series of high mannose-type oligosaccharides, which were not detected in erythrocyte N-glycans of normal ***cells*** or of other HEMPAS cases: The former contains polylactosaminoglycans and the latter contains hybrid-type oligosaccharides. Keratansulfate (sulfated polylactosamines) in this patient's serum was abnormally low. The galactosyltransferase activity in microsomal membranes prepared from G.K.'s mononucleated ***cells*** was 24% of the normal level, whereas this enzyme activity in G.K.'s serum was comparatively higher than normal. Western blotting of G.K.'s membranes using antigalactosyltransferase antibodies showed that G.K. has reduced amounts of this enzyme present. The results collectively suggest that variant G.K. is defective in polylactosamine synthesis owing to the decreased quantity of the membrane-bound form of galactosyltransferase.

L5 ANSWER 93 OF 206 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 89016407 EMBASE <<LOGINID::20070409>>
DN 1989016407
TI Regulation of glycosylation. The influence of protein structure on N-linked oligosaccharide processing.
AU Hubbard S.C.
CS Center for Cancer Research, Massachusetts Institute of Technology, Cambridge, MA 02139, United States
SO Journal of Biological Chemistry, (1988) Vol. 263, No. 36, pp. 19303-19317. .
ISSN: 0021-9258 CODEN: JBCHA3
CY United States
DT Journal
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 12 Dec 1991
Last Updated on STN: 12 Dec 1991
AB The Sindbis virus glycoproteins, E1 and E2, comprise a useful model system for evaluating the effects of local protein structure on the processing of N-linked oligosaccharides by Golgi enzymes. The conversion of oligomannose to N-acetyllactosamine (complex) oligosaccharides is hindered to different extents at the four glycosylation sites, so that the complex/oligomannose ratio decreases in the order E1-Asn139 > E2-Asn196 > E1-Asn245 > E2-Asn318. The processing steps most susceptible to interference were deduced from the oligosaccharide compositions at hindered sites in virus from baby hamster kidney ***cells*** (BHK), chick embryo fibroblasts (CEF), and normal and hamster sarcoma virus (HSV)-transformed hamster fibroblasts (Nil-8). Persistence of Man6-9GlcNAc2 was taken to indicate interference with .alpha.2-mannosidase(s) I (.alpha.-mannosidase I), Man5GlcNAc2, with UDP-GlcNAc:.alpha.-D-mannoside .beta.1.fwdarw.2-N-acetylglucosaminyltransferase I (GlcNAc transferase I), and unbisected hybrid glycans, with GlcNAc transferase I-dependent .alpha.3(.alpha.6)-mannosidase (.alpha.-mannosidase II). Taken together, the results indicate that all four sites acquire a precursor oligosaccharide with equally high efficiency, but .alpha.-mannosidase I, GlcNAc transferase I, and .alpha.-mannosidase II are all impeded at E2-Asn318 and, to a lesser extent, at E1-Asn245. In contrast, sialic acid and galactose transfer to hybrid glycans (in BHK ***cells***) is virtually quantitative even at E2-Asn318. E2-Asn318 carried no complex oligosaccharides, but the structures of those at E1-Asn245 indicate almost complete GlcNAc transfer by UDP-GlcNAc:.alpha.-D-mannoside .beta.1.fwdarw.2-N- ***acetylglucosaminyltransferase*** ***II*** (GlcNAc transferase ***II***), galactosylation, and sialylation. Because the E2-Asn318 and E1-Asn245 glycans have previously been shown to be less accessible to a

steric probe than those at E2-Asn196 or E1-Asn139, a simple explanation for these results would be that .alpha.-mannosidase I, GlcNAc transferase I, and .alpha.-mannosidase II are more susceptible to steric hindrance than are the later processing steps examined. Finally, in addition to these site-specific effects, the overall extent of viral oligosaccharide processing varied with host and ***cellular*** growth status. For example, .alpha.-mannosidase I processing is more complete in BHK ***cells*** compared to CEF, and in confluent Nil-8 ***cells*** compared to subconfluent or HSV-transformed Nil-8 ***cells*** .

L5 ANSWER 94 OF 206 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 88275748 EMBASE <<LOGINID::20070409>>
DN 1988275748
TI .beta.-1,4-mannosyl-glycoprotein .beta.-1,4-N-***acetylglucosaminyltransferase*** ***III*** activity in human B and T lymphocyte lines and in tonsillar B and T lymphocytes.
AU Narasimhan S.; Lee J.W.W.; Cheung R.K.; Gelfand E.W.; Schachter H.
CS Division of Biochemistry, Research Institute, The Hospital for Sick Children, Toronto, Ont. M5G 1X8, Canada
SO Biochemistry and Cell Biology, (1988) Vol. 66, No. 8, pp. 889-900. . ISSN: 0829-8211 CODEN: BCBIEQ
CY Canada
DT Journal
FS 026 Immunology, Serology and Transplantation
029 Clinical Biochemistry
LA English
SL French
ED Entered STN: 11 Dec 1991
Last Updated on STN: 11 Dec 1991
DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

L5 ANSWER 95 OF 206 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 88161536 EMBASE <<LOGINID::20070409>>
DN 1988161536
TI Control of glycoprotein synthesis. Purification and characterization of rabbit liver UDP-N-acetylglucosamine:.alpha.-3-D-mannoside .beta.-1,2-N-acetylglucosaminyltransferase I.
AU Nishikawa Y.; Pegg W.; Paulsen H.; Schachter H.
CS Department of Biochemistry, Research Institute, The Hospital for Sick Children, Toronto, Ont. M5G 1X8, Canada
SO Journal of Biological Chemistry, (1988) Vol. 263, No. 17, pp. 8270-8281. . ISSN: 0021-9258 CODEN: JBCHA3
CY United States
DT Journal
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 11 Dec 1991
Last Updated on STN: 11 Dec 1991
AB UDP-N-acetylglucosamine:.alpha.-3-D-mannoside .beta.-1,2-N-acetylglucosaminyltransferase I catalyzes an essential first step in the conversion of high mannose to hybrid and complex N-glycans (Schachter, H. (1986) Biochem. ***Cell*** Biol. 64, 163-181; Oppenheimer, C.L., and Hill, R.L. (1981) J. Biol. Chem. 256, 799-804), i.e. the addition of GlcNAc to (Man.alpha.1-6(Man.alpha.1-3)Man.alpha.1-6)(Man.alpha.1-3)Man.beta.1-4GlcNAc-OR to form (Man.alpha.1-6(Man.alpha.1-3)Man.alpha.1-6)(GlcNAc.beta.1-2Man.alpha.1-3)Man.beta.1-4GlcNAc-OR. The enzyme has been purified from Triton X-100 extracts of rabbit liver by chromatography on CM-Sephadex, Affi-Gel blue, UDP-hexanolamine-Sepharose, and a novel adsorbent in which UDP-GlcNAc is linked to thiopropyl-Sepharose at the 5-position of uracil. The enzyme exists in crude liver extracts in two molecular weight forms separable on Sephadex G-200. The low molecular weight form was purified 64,000-fold with a specific activity of 19.8 .mu.mol/min/mg. The pure enzyme was free of N-***acetylglucosaminyltransferase*** ***II*** -V activities. Sodium dodecyl sulfate-polyacrylamide gel electrophoresis showed a single major band of M(r) 45,000 and two minor bands of M(r) 54,000 and 50,000. All three bands showed retarded elution from an affinity column in which the acceptor substrate for the transferase was covalently linked to Sepharose. Kinetic analysis indicated a largely ordered sequential mechanism with UDP-GlcNAc binding to the enzyme first and UDP leaving last. Studies with synthetic analogues of the substrate Man.alpha.1-6(Man.alpha.1-3)Man.beta.1-4GlcNAc showed that an unsubstituted equatorial hydroxyl on carbon 4 of the .beta.-linked Man residue was essential for enzyme activity.

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AN 88131238 EMBASE <<LOGINID::20070409>>
DN 1988131238
TI A method for the determination of N- ***acetylglucosaminyltransferase*** ***III*** activity in rat tissues involving HPLC.
AU Nishikawa A.; Fujii S.; Sugiyama T.; Taniguchi N.
CS Department of Biochemistry, Osaka University Medical School, Nakanoshima, Osaka 530, Japan
SO Analytical Biochemistry, (1988) Vol. 170, No. 2, pp. 349-354. . ISSN: 0003-2697 CODEN: ANBCA2
CY United States
DT Journal
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 11 Dec 1991
Last Updated on STN: 11 Dec 1991
AB A fluorescence assay method for UDP-GlcNAc:glycopeptide .beta.1-4 N-***acetylglucosaminyltransferase*** (Gn-T- ***III***) has been developed involving a pyridylaminated sugar as a substrate. A fluorescent sugar chain, in which the reducing end of the GlcNAc.beta.1-2Man.alpha.1-6(GlcNAc.beta.1-2Man.alpha.1-3)Man.beta.1-4GlcNAc.beta.1-4GlcNAc has been aminated with 2-aminopyridine, is incubated with an enzyme sample, and then the fluorescent product with a bisecting N-acetylglucosamine is separated by reverse-phase high performance liquid chromatography and quantitated with a fluorescence detector. This assay method was found to be sensitive enough for the detection of 0.1 pmol of a reaction product. This assay is a reliable alternative to the use of a radiolabeled substrate and can be used for assaying N-acetylglucosaminyltransferase activity in crude extracts of various rat tissues. The kinetic experiments were carried out using crude enzyme extracts from the rat kidney. The enzyme has a pH optimum of 6.25 and requires Mn2+. The K(m) values for UDP-GlcNAc and a sugar acceptor substrate were found to be 3.1 mM and 190 .mu.M, respectively. The enzyme activity in the rat kidney was higher than those in the other tissues examined.

L5 ANSWER 97 OF 206 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 88122903 EMBASE
=> d his

(FILE 'HOME' ENTERED AT 18:33:16 ON 09 APR 2007)

FILE 'EMBASE, BIOSIS, CAPLUS' ENTERED AT 18:33:25 ON 09 APR 2007
L1 3949 S ACETYLGLUCOSAMINYLTRANSFERASE
L2 682 S L1 (3A) (II OR III)
L3 485 S L2 AND CELL?
L4 271 DUP REM L3 (214 DUPLICATES REMOVED)
L5 206 S L4 AND PY<=2003

=> s l3 and (yeast or insect or plant
UNMATCHED LEFT PARENTHESIS 'AND (YEAST'
The number of right parentheses in a query must be equal to the number of left parentheses.

=> s l3 and (yeast or insect or plant)
L6 55 L3 AND (YEAST OR INSECT OR PLANT)

=> dup rem l6
PROCESSING COMPLETED FOR L6
L7 36 DUP REM L6 (19 DUPLICATES REMOVED)

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 36 ANSWERS - CONTINUE? Y/(N):y

L7 ANSWER 1 OF 36 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2007:114069 CAPLUS <<LOGINID::20070409>>
DN 146:212635
TI Remodeling and glycoconjugation of erythropoietin and other therapeutic polypeptides
IN Defrees, Shawn; Zopf, David A.; Bayer, Robert J.; Hakes, David James; Bowe, Caryn; Chen, Xi
PA USA
SO U.S. Pat. Appl. Publ., 753pp., Cont.-in-part of U.S. Ser. No. 410,945. CODEN: USXXCO
DT Patent
LA English
FAN.CNT 17

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2007027068	A1	20070201	US 2005-530972	20051205
WO 2003031464	A2	20030417	WO 2002-US32263	20021009
WO 2003031464	A3	20060302		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 2004137557	A1	20040715	US 2002-287994	20021105
US 7138371	B2	20061121		
US 2007042458	A1	20070222	US 2003-410945	20030409
WO 2004033651	A2	20040422	WO 2003-US31974	20031008
WO 2004033651	A3	20060330		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES,

FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRAI US 2001-328523P P 20011010
US 2001-344692P P 20011019
US 2001-334233P P 20011128
US 2001-334301P P 20011128
US 2002-387292P P 20020607
US 2002-391777P P 20020625
US 2002-396594P P 20020717
US 2002-404249P P 20020816
US 2002-407527P P 20020828
WO 2002-US32263 A1 20021009
US 2002-287994 A2 20021105
US 2003-360770 B2 20030106
US 2003-360779 B2 20030219
US 2003-410945 A2 20030409
WO 2003-US31974 W 20031008

AB The invention includes methods and compns. for remodeling a peptide mol., including the addn. or deletion of one or more glycosyl groups to a peptide, and/or the addn. of a modifying group to a peptide. A key feature of the invention is to take a peptide produced by any ***cell*** type and generate a core glycan structure on the peptide, following which the glycan structure is then remodeled in vitro to generate a peptide having a glycosylation pattern suitable for therapeutic use in a mammal. The invention includes remodeling and PEGylation of erythropoietin, for use in treating anemia or kidney dialysis patients.

L7 ANSWER 2 OF 36 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

AN 2007114309 EMBASE <<LOGINID::20070409>>

TI Efficient introduction of a bisecting GlcNAc residue in tobacco N-glycans by expression of the gene encoding human N- ***acetylglucosaminyltransfer*** *** ase*** ***III*** .

AU Rouwendal G.J.A.; Wuhrer M.; Florack D.E.A.; Koeleman C.A.M.; Deelder A.M.; Bakker H.; Stoopen G.M.; van Die I.; Helsper J.P.F.G.; Hokke C.H.; Bosch D.

CS G.J.A. Rouwendal, Business Unit Bioscience, Plant Research International B.V., Wageningen University and Research Centre, Droevendaalsesteeg 1, 6708 PB Wageningen, Netherlands. gerard.rouwendal@wur.nl

SO Glycobiology, (2007) Vol. 17, No. 3, pp. 334-344. .
Refs: 49
ISSN: 0959-6658 E-ISSN: 1460-2423 CODEN: GLYCE3

CY United Kingdom

DT Journal; Article

FS 029 Clinical Biochemistry
037 Drug Literature Index

LA English

SL English

ED Entered STN: 27 Mar 2007
Last Updated on STN: 27 Mar 2007

AB In this study, we show that introduction of human N- ***acetylglucosaminyltransferase*** (GnT)- ***III*** gene into tobacco plants leads to highly efficient synthesis of bisected N-glycans. Enzymatically released N-glycans from leaf glycoproteins of wild-type and transgenic GnT-III plants were profiled by matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF-MS) in native form. After labeling with 2-aminobenzamide, profiling was performed using normal-phase high-performance liquid chromatography with fluorescence detection, and glycans were structurally characterized by MALDI-TOF/TOF-MS and reverse-phase nano-liquid chromatography-MS/MS. These analyses revealed that most of the complex-type N-glycans in the plants expressing GnT-III were bisected and carried at least two terminal N-acetylglucosamine (GlcNAc) residues in contrast to wild-type plants, where a considerable proportion of N-glycans did not contain GlcNAc residues at the nonreducing end. Moreover, we have shown that the majority of N-glycans of an antibody produced in a ***plant*** expressing GnT-III is also bisected. This might improve the efficacy of therapeutic antibodies produced in this type of transgenic ***plant*** . .COPYRGT. 2007 Oxford University Press.

L7 ANSWER 3 OF 36 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN DUPLICATE 1

AN 2006242628 EMBASE <<LOGINID::20070409>>

TI Influence of variable N-glycosylation on the cytolytic potential of chimeric CD19 antibodies.

AU Barbin K.; Stieglmaier J.; Saul D.; Stieglmaier K.; Stockmeyer B.; Pfeiffer M.; Lang P.; Fey G.H.

CS Dr. G.H. Fey, Department of Genetics, University of Erlangen-Nuremberg, Staudtstrasse 5, D 91058 Erlangen, Germany. gfey@biologie.uni-erlangen.de

SO Journal of Immunotherapy, (2006) Vol. 29, No. 2, pp. 122-133. .
Refs: 50
ISSN: 1524-9557 CODEN: JOIME7

PUI 000023712006030000002

CY United States

DT Journal; Article

FS 016 Cancer
025 Hematology
026 Immunology, Serology and Transplantation
037 Drug Literature Index

LA English

SL English

ED Entered STN: 22 Jun 2006
Last Updated on STN: 22 Jun 2006

AB To investigate the influence of N-linked oligosaccharides at asparagines-297 on the cytolytic potential of chimeric CD19 antibodies, three distinct variants were generated by production in different expression systems. The same chimeric CD19 antibody was produced in Sf21 ***insect*** ***cells*** , human 293 T ***cells*** , and 293 T ***cells*** expressing a co-transfected .beta.1,4-N- ***acetylglucosaminyltransferase*** ***III*** (GnTIII). The N-glycan structures and the cytolytic potential of the antibodies produced in these three systems were directly compared. After expression in ***insect*** ***cells*** , the antibody carried paucimannosidic N-linked oligosaccharides, distinct from the complex biantennary carbohydrate moieties attached to the product from human ***cells*** . After co-expression with GnTIII in human ***cells*** , the antibody carried an eightfold greater percentage of oligosaccharides with a bisecting N-acetylglucosamine (78.7% versus 9.6%) and a 30-fold increased proportion of bisecting, defucosylated oligosaccharides (15.9% versus 0.5%). The ***insect*** ***cell*** product triggered stronger antibody-dependent ***cellular*** cytotoxicity (ADCC) of a human leukemia-derived ***cell*** line than the product from non-re-engineered 293 T ***cells*** and was equally effective at 50- to 100-fold lower concentrations. The antibody from glyco-engineered 293 T ***cells*** had comparable lytic activity as the ***insect*** ***cell*** product. Both mediated significant ADCC at lower effector-to-target ***cell*** ratios than the antibody from non-re-engineered 293 T ***cells*** , and both were highly effective against primary blasts from pediatric leukemia patients. The data demonstrate the influence of the N-glycosylation pattern on the ADCC activity of chimeric CD19 antibodies and point to the importance of suitable expression systems for the production of highly active therapeutic antibodies. Copyright .COPYRGT. 2006 by Lippincott Williams & Wilkins.

L7 ANSWER 4 OF 36 CAPLUS COPYRIGHT 2007 ACS on STN

AN 2005:1154698 CAPLUS <<LOGINID::20070409>>

DN 143:433718

TI Genetically engineered ***yeast*** for production of human-like glycoproteins with terminal galactose residues

IN Davidson, Robert; Gerngross, Tillman; Wildt, Stefan; Choi, Byung-Kwon; Nett, Juergen; Bobrowicz, Piotr; Hamilton, Stephen

PA Glycofi, Inc., USA

SO PCT Int. Appl., 120 pp.
CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 25

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2005100584	A2	20051027	WO 2005-IB51249	20050415
WO 2005100584	A3	20061221		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2005233387	A1	20051027	AU 2005-233387	20050415
CA 2562772	A1	20051027	CA 2005-2562772	20050415
EP 1737969	A2	20070103	EP 2005-732293	20050415
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, LV, MK, YU				

PRAI US 2004-562424P P 20040415
WO 2005-IB51249 W 20050415

AB The invention provides a lower eukaryotic host ***cell*** producing human-like glycoproteins characterized as having a terminal .beta.-galactose residue and essentially lacking fucose and sialic acid residues. The invention also provides methods and compns., including genetic vectors, for catalyzing the transfer of a galactose residue from UDP-galactose onto an acceptor substrate in a recombinant lower eukaryotic host ***cell*** . In addn. to a UDP-Gal:.beta.GlcNAc .beta.-1,4-galactosyltransferase, expression of UDP-galactose transporter(s), a UDP-specific diphosphatase, and UDP-galactose-4-epimerase, galactokinase, or galactose-1-phosphate uridyltransferase activities allow transfer of galactose residues onto preferred acceptor substrates for use as therapeutic glycoproteins. The invention claims polypeptide sequences for gene galE UDP-galactose C4 epimerase enzyme and conserved motifs. Methods of the invention can be applied to therapeutic glycoproteins such as erythropoietin, cytokines, blood coagulation factors, Igs, growth factors, or plasminogen. The examples provide maps of integrating plasmid vectors encoding human GalTI, S. pombe gene galE epimerase, and D. melanogaster gene UGT UDP-galactose transporter. The secreted kringle 3 (K3) domain of plasminogen was the reporter protein for glycosylation in transformed Pichia pastoris strains. N-linked glycans obtained from K3 were analyzed by MALDI-TOF mass spectrometry. A P. pastoris strain with och1 and alg3 gene deletions, active fusion constructs of mouse mannosidase IB and human GnTI, the Kluyveromyces

lactis UDP-GlcNAc transporter gene, and a human GalTI gene leader fusion construct had approx. 10-20% of GlcNAc2Man3GlcNAc2 N-glycans on K3 converted to GalGlcNAc2Man3GlcNAc2 and 1-2% to Gal2GlcNAc2Man3GlcNAc2.
When a strain with the same genotype was also transformed with the Saccharomyces cerevisiae epimerase gene GAL10 under control of the PMAI promoter, about 2/3 of the N-glycans released from K3 contained an addnl. hexose residue (HexGlcNAcMan5GlcNAc2) that could be removed by sol. .beta.-1,4-galactosidase.

L7 ANSWER 5 OF 36 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2005:409684 CAPLUS <<LOGINID::20070409>>
DN 142:458111
TI Production of human glycosylated proteins in transgenic insects
IN Jarvis, Donald; Van Beek, Nikolai; Fraser, Malcolm
PA Chesapeake Perl, Inc., USA
SO PCT Int. Appl., 81 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2005042753	A1	20050512	WO 2004-US35553	20041028
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2007067855	A1	20070322	US 2006-577528	20060428
PRAI US 2003-514741P	P	20031028		
WO 2004-US35553	W	20041028		

AB The invention provides transgenic insects, or progeny thereof, whose ***cells*** contain at least one integrated nucleic acid encoding two or more N-glycosylation enzymes that are used to glycosylate a heterologous protein with a mammalianized (humanized) pattern. Specifically, the invention provides transgenic insects transformed with vectors encoding: (a) various N-acetylglucosaminyltransferases (GlcNAc-Ts), sialyltransferases (.alpha.2,6-sialyltransferase and .alpha.2,3-sialyltransferase), sialic acid synthase and CMP-sialic acid synthetase; (b) various auxiliary glycosylation proteins (such as transport proteins); and (c) a heterologous protein of interest (such as antibody, receptor, vaccine). The invention relates that said glycosylation enzymes are expressed and used to produce glycosylated proteins of interest. The invention also provides methods for producing said humanized glycosylated proteins using transgenic ***insect*** larva and baculovirus-based or transposon-based vectors carrying said nucleic acids. The invention further provides a library of different types of TRANSPILLAR larva expressing different glycoproteins of interest. The invention briefly discussed the use of said transgenic ***insect*** ***cells*** in manufg. authentic human-type glycoproteins for therapeutic applications (no data).
RE.CNT 10 THERE ARE 10 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 6 OF 36 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2005:1028015 CAPLUS <<LOGINID::20070409>>
DN 143:300313
TI N-acetylglucosamintransferase III expression in genetically modified lower eukaryotes
IN Bobrowicz, Piotr; Hamilton, Stephen R.; Gerngross, Tilman U.; Wildt, Stefan; Choi, Byung-Kwon; Nett, Juergen Hermann; Davidson, Robert C.
PA USA
SO U.S. Pat. Appl. Publ., 163 pp., Cont.-in-part of U.S. Ser. No. 371,877.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 25

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2005208617	A1	20050922	US 2003-680963	20031007
US 2002137134	A1	20020926	US 2001-892591	20010627
US 7029872	B2	20060418		
EP 1522590	A1	20050413	EP 2004-25648	20010627
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY, TR				
WO 2003056914	A1	20030717	WO 2002-US41510	20021224
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
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US 2004018590	A1	20040129	US 2003-371877	20030220
AU 2004213859	A1	20040902	AU 2004-213859	20040220
AU 2004213868	A1	20040902	AU 2004-213868	20040220
CA 2516520	A1	20040902	CA 2004-2516520	20040220
CA 2516550	A1	20040902	CA 2004-2516550	20040220
WO 2004074458	A2	20040902	WO 2004-US5128	20040220
WO 2004074458	A3	20041229		
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WO 2004074461	A2	20040902	WO 2004-US5191	20040220
WO 2004074461	A3	20050317		
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RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
EP 1597381	A2	20051123	EP 2004-713388	20040220
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
EP 1599595	A2	20051130	EP 2004-713412	20040220
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
JP 2006518597	T	20060817	JP 2006-503757	20040220
JP 2006518600	T	20060817	JP 2006-503776	20040220
US 2007037248	A1	20070215	US 2006-546101	20060803
PRAI US 2000-214358P	P	20000628		
US 2000-215638P	P	20000630		
US 2001-279997P	P	20010330		
US 2001-892591	A2	20010627		
US 2001-344169P	P	20011227		
WO 2002-US41510	A2	20021224		
US 2003-371877	A2	20030220		
EP 2001-954606	A3	20010627		
WO 2002-US241510	W	20021224		
US 2003-680963	A	20031007		
WO 2004-US5128	A	20040220		
WO 2004-US5191	A	20040220		
US 2005-500240	A2	20050323		

AB The present invention relates to eukaryotic host ***cells*** having modified oligosaccharides which may be modified further by heterologous expression of a set of glycosyltransferases, sugar transporters and mannosidases to become host-strains for the prodn. of mammalian, e.g., human therapeutic glycoproteins. The present invention relates to methods and comps. by which non-human eucaryotic ***cells*** , such as fungi or other eukaryotic ***cells*** , can be genetically modified to produce glycosylated proteins (glycoproteins) having patterns of glycosylation similar to those of glycoproteins produced by animal ***cells*** , esp. human ***cells*** , which are useful as human or animal therapeutic agents. The process provides an engineered host ***cell*** which can be used to express and target any desirable gene(s) involved in glycosylation. Host ***cells*** with modified lipid-linked oligosaccharides are created or selected. N-glycans made in the engineered host ***cells*** exhibit GnTIII activity, which produce bisected N-glycan structures and may be modified further by heterologous expression of one or more enzymes, e.g., glycosyltransferases, sugar transporters and mannosidases, to yield human-like glycoproteins. For the prodn. of therapeutic proteins, this method may be adapted to engineer ***cell*** lines in which any desired glycosylation structure may be obtained.

L7 ANSWER 7 OF 36 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2005:409140 CAPLUS <<LOGINID::20070409>>
DN 142:487367
TI ***Cell*** -free in vitro glycan remodeling and enzymic glycoconjugation of Factor IX for treating hemophilia B
IN Defrees, Shawn; Zopf, David; Bayer, Robert; Bowe, Caryn; Hakes, David; Chen, Xi
PA Neose Technologies, Inc., USA
SO U.S. Pat. Appl. Publ., 761 pp., Cont.-in-part of U. S. Ser. No. 360,779.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 17

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2005100982	A1	20050512	US 2003-410897	20030409
US 7179617	B2	20070220		
WO 2003031464	A2	20030417	WO 2002-US32263	20021009
WO 2003031464	A3	20060302		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH,				

PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PI US 2004137557 A1 20040715 US 2002-287994 20021105
US 7138371 B2 20061121
AU 2004236174 A1 20041118 AU 2004-236174 20040409
CA 2522345 A1 20041118 CA 2004-2522345 20040409
WO 2004099231 A2 20041118 WO 2004-US11494 20040409
WO 2004099231 A3 20060316
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
EP 1615945 A2 20060118 EP 2004-750118 20040409
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, HR
BR 2004009277 A 20060321 BR 2004-9277 20040409
CN 1863458 A 20061115 CN 2004-80015918 20040409
US 2007026485 A1 20070201 US 2006-552896 20060608
PRAI US 2001-328523P P 20011010
US 2001-344692P P 20011019
US 2001-334233P P 20011128
US 2001-334301P P 20011128
US 2002-387292P P 20020607
US 2002-391777P P 20020625
US 2002-396594P P 20020717
US 2002-404249P P 20020816
US 2002-407527P P 20020828
WO 2002-US32263 A1 20021009
US 2002-287994 A2 20021105
US 2003-360770 A2 20030106
US 2003-360779 A2 20030219
US 2003-410897 A 20030409
US 2003-410913 A 20030409
US 2003-410930 A 20030409
US 2003-410945 A 20030409
US 2003-410962 A 20030409
US 2003-410980 A 20030409
US 2003-410997 A 20030409
US 2003-411012 A 20030409
US 2003-411026 A 20030409
US 2003-411037 A 20030409
US 2003-411043 A 20030409
US 2003-411044 A 20030409
US 2003-411049 A 20030409
WO 2004-US11494 A 20040409

AB A method is disclosed for remodeling a peptide, including the addn. or deletion, if necessary, of one or more glycosyl groups of the peptide, then enzyme-mediated attachment of a PEGylated sugar. A key feature of the invention is to take a peptide produced by any ***cell*** type and generate a core glycan structure on the peptide, following which the glycan structure is then remodeled in vitro to generate a peptide having a glycosylation pattern suitable for therapeutic use in a mammal. The invention includes a ***cell*** -free, in vitro method of remodeling and PEGylation of Factor IX using glycosyltransferase, sialyltransferase and sialidase. Exemplary glycoPEGylation of Factor IX produced in CHO ***cells*** , direct sialyl-glycoPEGylation of Factor IX, and sialic acid capping of glycoPEGylated Factor IX are described. Other proteins were glycoPEGylated in a similar manner. The Factor IX of the invention is used for treating hemophilia B in human.

RE.CNT 192 THERE ARE 192 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 8 OF 36 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2005:122585 CAPLUS <<LOGINID::20070409>>
DN 142:217398
TI ***Cell*** -free in vitro glycoconjugation of interleukin 2 as therapeutic agent against cancer and AIDS in mammal and human
IN Defrees, Shawn; Zopf, David; Bayer, Robert; Bowe, Caryn; Hakes, David; Chen, Xi
PA Neose Technologies, Inc., USA
SO U.S. Pat. Appl. Publ., 750 pp., Cont.-in-part of U.S. Ser. No. 360,779.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 17

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2005031584	A1	20050210	US 2003-410980	20030409
WO 2003031464	A2	20030417	WO 2002-US32263	20021009
WO 2003031464	A3	20060302		

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RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 2004137557 A1 20040715 US 2002-287994 20021105
US 7138371 B2 20061121
AU 2004236174 A1 20041118 AU 2004-236174 20040409
CA 2522345 A1 20041118 CA 2004-2522345 20040409
WO 2004099231 A2 20041118 WO 2004-US11494 20040409
WO 2004099231 A3 20060316
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
EP 1615945 A2 20060118 EP 2004-750118 20040409
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, HR
CN 1863458 A 20061115 CN 2004-80015918 20040409
US 2007026485 A1 20070201 US 2006-552896 20060608
PRAI US 2001-328523P P 20011010
US 2001-344692P P 20011019
US 2001-334233P P 20011128
US 2001-334301P P 20011128
US 2002-387292P P 20020607
US 2002-391777P P 20020625
US 2002-396594P P 20020717
US 2002-404249P P 20020816
US 2002-407527P P 20020828
WO 2002-US32263 A1 20021009
US 2002-287994 A2 20021105
US 2003-360770 A2 20030106
US 2003-360779 A2 20030219
US 2003-410897 A 20030409
US 2003-410913 A 20030409
US 2003-410930 A 20030409
US 2003-410945 A 20030409
US 2003-410962 A 20030409
US 2003-410980 A 20030409
US 2003-410997 A 20030409
US 2003-411012 A 20030409
US 2003-411026 A 20030409
US 2003-411037 A 20030409
US 2003-411043 A 20030409
US 2003-411044 A 20030409
US 2003-411049 A 20030409
WO 2004-US11494 A 20040409

AB The invention includes methods and compns. for remodeling a peptide mol., including the addn. or deletion of one or more glycosyl groups to a peptide, and/or the addn. of a modifying group to a peptide. The method uses enzyme to remove or add phosphate, sulfate, carboxylate and/or ester group-contg. saccharide to interleukin 2 peptide, and then conjugate the saccharide-linked interleukin 2 with modifying group such as polymer, therapeutic moiety, detectable label, toxin, radioisotope, targeting moiety and peptide. The saccharide group comprises monosaccharyl, oligosaccharyl, glycosyl, truncated glycan, mannosyl, GlcNAc, xylosyl, sialyl, galactosyl, glucosyl or GalNAc. The enzyme for the saccharide addn. or removal is a prokaryotic or eukaryotic glycosyltransferase selected from sialyltransferase, galactosyltransferase, glucosyltransferase, GalNAc transferase, GlcNAc transferase, fucosyltransferase, mannosyltransferase, endo-N-acetylgalactosaminidase, glycosidase, sialidase, mannosidase, etc. The substrate is a nucleotide sugar such as UDP-glucose, UDP-galactose, UDP-galactosamine, UDP-glucosamine, UDP-N-acetylgalactosamine, UDP-N-acetylglucosamine, GDP-mannose, GDP-fucose, CMP-sialic acid and CMP-NeuAc. .

L7 ANSWER 9 OF 36 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
DUPLICATE 2
AN 2006:125729 BIOSIS <<LOGINID::20070409>>
DN PREV200600113050
TI Control of recombinant monoclonal antibody effector functions by Fc N-glycan remodeling in vitro.
AU Hodoniczky, Jason; Zheng, Yuan Zhi; James, David C. [Reprint Author]
CS Univ Queensland, Sch Engn, St Lucia, Qld 4072, Australia
davidj@cheque.uq.edu.au
SO Biotechnology Progress, (NOV-DEC 2005) Vol. 21, No. 6, pp. 1644-1652.
CODEN: BIPRET. ISSN: 8756-7938.
DT Article
LA English
ED Entered STN: 15 Feb 2006
Last Updated on STN: 15 Feb 2006

AB N-Glycans at Asn(297) in the Fc domain of IgG molecules are required for Fc receptor-mediated effector functions such as antibody-dependent ***cell*** -mediated cytotoxicity (ADCC) and complement-dependent cytotoxicity (CDC). In this study we have specifically remodeled the Fc N-glycans of intact recombinant IgG(1) therapeutic monoclonal antibody (Mab) products, Rituxan and Herceptin, with a soluble recombinant rat beta-1,4-N- ***acetylglucosaminyltransferase*** ***III*** (rGnTIII) produced by baculovirus-infected ***insect*** ***cells*** . N-Glycan remodeling in vitro permitted a controlled and selective transfer of a bisecting beta 1,4-linked GlcNAc to the core beta-linked mannose of degalactosylated Mab N-glycans to yield Mabs varying in bisecting GlcNAc: content from 31% to 85%. This was confirmed by analysis of N-glycans by both normal phase HPLC and MALDI-MS, the latter yielding the expected mass increase of 203.2 Da with no other oligosaccharide modifications evident. ADCC of remodeled Rituxan and Herceptin Mabs was determined using peripheral blood mononuclear ***cells*** as effectors and either CD20(+) (SKW6.4 and SU-DHL-4) or Her2(+) (SKBR-3) target ***cells*** , respectively. A conserved 10-fold increase in ADCC was observed for both remodeled therapeutic Mabs with high (> 80%) bisecting GlcNAc content. In contrast, although the presence of a bisecting GlcNAc had minimal effect on CDC, degalactosylation of Rituxan reduced CDC by approximately half, relative to unmodified (variably galactosylated) control Mab. In summary, our data suggests that in vitro remodeling of therapeutic Mab Fc N-glycans may be utilized to control the therapeutic efficacy of Mabs in vivo and to offer a more "humanized" glycoform profile for recombinant Mab products.

L7 ANSWER 10 OF 36 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2005:465641 CAPLUS <<LOGINID::20070409>>
DN 143:189035
TI Arabidopsis thaliana .beta.1,2-xylosyltransferase: an unusual glycosyltransferase with the potential to act at multiple stages of the ***plant*** N-glycosylation pathway
AU Bencur, Peter; Steinkellner, Herta; Svoboda, Barbara; Mucha, Jan; Strasser, Richard; Kolarich, Daniel; Hann, Stephan; Koellensperger, Gunda; Gloessl, Josef; Altmann, Friedrich; Mach, Lukas
CS Department fuer Angewandte Pflanzenwissenschaften und Pflanzenbiotechnologie, Institut fuer Angewandte Genetik und Zellbiologie, Universitaet fuer Bodenkultur Wien, Vienna, A-1190, Austria
SO Biochemical Journal (2005), 388(2), 515-525
CODEN: BIJOAK; ISSN: 0264-6021
PB Portland Press Ltd.
DT Journal
LA English
AB XylT (.beta.1,2-xylosyltransferase) is a unique Golgi-bound glycosyltransferase that is involved in the biosynthesis of glycoprotein-bound N-glycans in plants. To delineate the catalytic domain of XylT, a series of N-terminal deletion mutants was heterologously expressed in ***insect*** ***cells*** . Whereas the first 54 residues could be deleted without affecting the catalytic activity of the enzyme, removal of an addnl. five amino acids led to the formation of an inactive protein. Characterization of the N-glycosylation status of recombinant XylT revealed that all three potential N-glycosylation sites of the protein are occupied by N-linked oligosaccharides. However, an unglycosylated version of the enzyme displayed substantial catalytic activity, demonstrating that N-glycosylation is not essential for proper folding of XylT. In contrast with most other glycosyltransferases, XylT is enzymically active in the absence of added metal ions. This feature is not due to any metal ion directly assocd. with the enzyme. The precise acceptor substrate specificity of XylT was assessed with several physiol. relevant compds. and the xylosylated reaction products were subsequently tested as substrates of other Golgi-resident glycosyltransferases. These expts. revealed that the substrate specificity of XylT permits the enzyme to act at multiple stages of the ***plant*** N-glycosylation pathway.
RE.CNT 34 THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 11 OF 36 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:720587 CAPLUS <<LOGINID::20070409>>
DN 141:237748
TI N- ***acetylglucosaminyltransferase*** ***III*** and other N-glycan-processing enzymes expressed in lower eukaryotes for the biosynthesis of human-like oligosaccharide structures in glycoproteins
IN Bobrowicz, Piotr; Hamilton, Stephen R.; Gerngross, Tillman U.; Wildt, Stefan; Choi, Byung-Kwon; Nett, Juergen Hermann; Davidson, Robert C.
PA USA
SO PCT Int. Appl., 193 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 25

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2004074458	A2	20040902	WO 2004-US5128	20040220
WO 2004074458	A3	20041229		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2004018590	A1	20040129	US 2003-371877	20030220
US 2005208617	A1	20050922	US 2003-680963	20031007
AU 2004213859	A1	20040902	AU 2004-213859	20040220
CA 2516520	A1	20040902	CA 2004-2516520	20040220
EP 1599595	A2	20051130	EP 2004-713412	20040220
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
JP 2006518597	T	20060817	JP 2006-503757	20040220
PRAI US 2003-371877	A	20030220		
US 2003-680963	A	20031007		
US 2000-214358P	P	20000628		
US 2000-215638P	P	20000630		
US 2001-279997P	P	20010330		
US 2001-892591	A2	20010627		
US 2001-344169P	P	20011227		
WO 2002-US41510	A2	20021224		
WO 2004-US5128	A	20040220		

AB The present invention relates to eukaryotic host ***cells*** having modified oligosaccharides which may be modified further by heterologous expression of a set of glycosyltransferases, sugar transporters, and mannosidases to become host-strains for the prodn. of mammalian, e.g., human therapeutic glycoproteins. The process provides an engineered host ***cell*** such as Pichia pastoris which can be used to express and target any desirable gene(s) involved in glycosylation. Host ***cells*** with modified lipid-linked oligosaccharides are created or selected. N-glycans made in the engineered host ***cells*** exhibit N- ***acetylglucosaminyltransferase*** ***III*** (GnTIII) activity, which produce bisected N-glycan structures and may be modified further by heterologous expression of one or more enzymes, e.g., glycosyltransferases, sugar transporters and mannosidases, to yield human-like glycoproteins. For the prodn. of therapeutic proteins, this method may be adapted to engineer ***cell*** lines in which any desired glycosylation structure may be obtained.

L7 ANSWER 12 OF 36 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:634026 CAPLUS <<LOGINID::20070409>>
DN 141:172878
TI Engineering of glycosylation profile of antibody Fc region to increase Fc receptor binding affinity and effector function for treating cancer
IN Umana, Pablo; Bruenker, Peter; Ferrara, Claudia; Suter, Tobias
PA Glycart Biotechnology Ag, Switz.
SO PCT Int. Appl., 231 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2004065540	A2	20040805	WO 2004-IB844	20040122
WO 2004065540	A3	20050324		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI				
AU 2004205802	A1	20040805	AU 2004-205802	20040122
CA 2513797	A1	20040805	CA 2004-2513797	20040122
US 2004241817	A1	20041202	US 2004-761435	20040122
EP 1587921	A2	20051026	EP 2004-704310	20040122
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
CN 1761746	A	20060419	CN 2004-80007564	20040122
JP 2006516893	T	20060713	JP 2006-500338	20040122
IN 2005KN01628	A	20060901	IN 2005-KN1628	20050816
NO 2005003872	A	20051021	NO 2005-3872	20050818
PRAI US 2003-441307P	P	20030122		
US 2003-491254P	P	20030731		
US 2003-495142P	P	20030815		
WO 2004-IB844	W	20040122		

AB The present invention relates to nucleic acid mols., including fusion constructs, having catalytic activity and the use of same in glycosylation engineering of host ***cells*** to generate polypeptides with improved therapeutic properties, including antibodies with increased Fc receptor binding and increased effector function. The engineered proteins or antibodies comprise Golgi localization domain of Golgi resident polypeptide such as .beta.(1,4)-N- ***acetylglucosaminyltransferase*** ***III*** , .beta.(1,4)-galactosyltransferase, mannosidase II, .beta.(1,2)-N-acetylglucosaminyltransferase I, .beta.(1,2)-N- ***acetylglucosaminyltransferase*** ***II*** , mannosidase I, .alpha.-mannosidase II, and .alpha.1-6 core fucosyltransferase. The effector function includes Fc-mediated ***cellular*** cytotoxicity of NK ***cells*** , macrophage, polymorphonuclear ***cells*** and monocytes; signaling of apoptosis induction; maturation of dendritic ***cells*** ; or T ***cell*** priming. The engineered antibodies include antibodies or humanized antibodies specific to human neuroblastoma, renal ***cell*** carcinoma, colon carcinoma, breast carcinoma, lung carcinoma, 17-1A antigen, CD20, CD22, CD30, CD40, PSMA, EGFR, PSCA, HLA-DR, MUC1, EpCAM, etc.

L7 ANSWER 13 OF 36 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:333839 CAPLUS <<LOGINID::20070409>>
DN 140:352406
TI Erythropoietin glycosylation and the modification of protein structure and activity for therapeutic use

IN De Frees, Shawn; Zopf, David; Bayer, Robert; Bowe, Caryn; Hakes, David; Chen, Xi
PA Neose Technologies, Inc., USA
SO PCT Int. Appl., 1018 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 17

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2004033651	A2	20040422	WO 2003-US31974	20031008
WO 2004033651	A3	20060330		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
WO 2003031464	A2	20030417	WO 2002-US32263	20021009
WO 2003031464	A3	20060302		
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RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2004137557	A1	20040715	US 2002-287994	20021105
US 7138371	B2	20061121		
US 2007042458	A1	20070222	US 2003-410945	20030409
CA 2501832	A1	20040422	CA 2003-2501832	20031008
AU 2003287035	A1	20040504	AU 2003-287035	20031008
BR 2003015178	A	20050816	BR 2003-15178	20031008
EP 1581622	A2	20051005	EP 2003-777555	20031008
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
JP 2006522738	T	20061005	JP 2005-501139	20031008
IN 2005KN00836	A	20060609	IN 2005-KN836	20050509
US 2007027068	A1	20070201	US 2005-530972	20051205
PRAI WO 2002-US32263	A	20021009		
US 2002-287994	A	20021105		
US 2003-360770	A	20030106		
US 2003-360779	A	20030219		
US 2003-410945	A	20030409		
US 2001-328523P	P	20011010		
US 2001-344692P	P	20011019		
US 2001-334233P	P	20011128		
US 2001-334301P	P	20011128		
US 2002-387292P	P	20020607		
US 2002-391777P	P	20020625		
US 2002-396594P	P	20020717		
US 2002-404249P	P	20020816		
US 2002-407527P	P	20020828		
WO 2003-US31974	W	20031008		

AB The invention includes methods and compns. for remodeling a peptide mol., including the addn. or deletion of one or more glycosyl groups to a peptide, and/or the addn. of a modifying group to a peptide. Methods of modifying the structure and properties of erythropoietin by introduction of glycosidation are described. The method uses substitution variants of erythropoietin to introduce sites that can be glycosylated enzymically. The primary glycosylation may then be used to add further sugar residues. The glycosidation, which may include the introduction of N-acetylglucose, N-acetylgalactose, and sialic acid and mannosyl and fucosyl oligosaccharides. The carbohydrate moiety may in turn be modified by PEGylation. A biantennary glycosidated deriv. of Epogen had 146% of the activity of the unmodified protein. The glycosylated proteins had longer serum half-lives than the unmodified protein and showed longer term effects on blood Hb levels.

L7 ANSWER 14 OF 36 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:80241 CAPLUS <<LOGINID::20070409>>
DN 140:158561
TI Combinatorial DNA library of mammalian glycosylation enzyme genes used for producing modified n-glycans in lower eukaryotes
IN Gerngross, Tillman U.; Wildt, Stefan; Choi, Byung-Kwon; Nett, Juergen Hermann; Bobrowicz, Piotr; Hamilton, Stephen R.; Davidson, Robert C.
PA USA
SO U.S. Pat. Appl. Publ., 97 pp., Cont.-in-part of U.S. Ser. No. 892,591.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 25

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2004018590	A1	20040129	US 2003-371877	20030220
US 2002137134	A1	20020926	US 2001-892591	20010627
US 7029872	B2	20060418		
EP 1522590	A1	20050413	EP 2004-25648	20010627
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY, TR				
US 2004230042	A1	20041118	US 2003-616082	20030708
US 2005208617	A1	20050922	US 2003-680963	20031007
US 2004171826	A1	20040902	US 2003-695243	20031027
AU 2004213859	A1	20040902	AU 2004-213859	20040220
AU 2004213860	A1	20040902	AU 2004-213860	20040220
AU 2004213861	A1	20040902	AU 2004-213861	20040220
AU 2004213868	A1	20040902	AU 2004-213868	20040220
AU 2004213869	A1	20040902	AU 2004-213869	20040220
CA 2516440	A1	20040902	CA 2004-2516440	20040220
CA 2516520	A1	20040902	CA 2004-2516520	20040220
CA 2516527	A1	20040902	CA 2004-2516527	20040220
CA 2516544	A1	20040902	CA 2004-2516544	20040220
CA 2516550	A1	20040902	CA 2004-2516550	20040220
WO 2004074458	A2	20040902	WO 2004-US5128	20040220
WO 2004074458	A3	20041229		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI				
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WO 2004074497	A2	20040902	WO 2004-US5131	20040220
WO 2004074497	A3	20041202		
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WO 2004074498	A2	20040902	WO 2004-US5132	20040220
WO 2004074498	A3	20050623		
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WO 2004074461	A2	20040902	WO 2004-US5191	20040220
WO 2004074461	A3	20050317		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
WO 2004074499	A2	20040902	WO 2004-US5244	20040220
WO 2004074499	A3	20050127		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
EP 1597379	A2	20051123	EP 2004-713369	20040220
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
EP 1597380	A2	20051123	EP 2004-713372	20040220
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
EP 1597381	A2	20051123	EP 2004-713388	20040220
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
EP 1599595	A2	20051130	EP 2004-713412	20040220
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
EP 1599596	A2	20051130	EP 2004-713437	20040220
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
JP 2006518597	T	20060817	JP 2006-503757	20040220
JP 2006518598	T	20060817	JP 2006-503759	20040220
JP 2006518599	T	20060817	JP 2006-503760	20040220
JP 2006518600	T	20060817	JP 2006-503776	20040220
JP 2006518601	T	20060817	JP 2006-503788	20040220
US 2006040353	A1	20060223	US 2005-108088	20050415
US 2006024304	A1	20060202	US 2005-187196	20050721
US 2006029604	A1	20060209	US 2005-187229	20050721
US 2006034828	A1	20060216	US 2005-187066	20050721
US 2006034830	A1	20060216	US 2005-187113	20050721
IN 2005KN01844	A	20061103	IN 2005-KN1844	20050916

US 2006078963 A1 20060413 US 2005-240432 20050930
US 2006177898 A1 20060810 US 2005-249061 20051011
US 2006148035 A1 20060706 US 2005-271235 20051110
US 2006257399 A1 20061116 US 2005-317191 20051222
US 2006286637 A1 20061221 US 2006-429672 20060505
US 2007037248 A1 20070215 US 2006-546101 20060803
PRAI US 2000-214358P P 20000628
US 2000-215638P P 20000630
US 2001-279997P P 20010330
US 2001-892591 A2 20010627
EP 2001-954606 A3 20010627
US 2001-344169P P 20011227
WO 2002-US241510 W 20021224
WO 2002-US41510 A2 20021224
US 2003-371877 A2 20030220
US 2003-616082 A 20030708
US 2003-680963 A 20031007
US 2003-695243 A 20031027
WO 2004-US5128 A 20040220
WO 2004-US5131 A 20040220
WO 2004-US5132 W 20040220
WO 2004-US5191 A 20040220
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US 2004-554139P P 20040317
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US 2004-589979P P 20040721
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US 2004-639541P P 20041223
US 2004-639542P P 20041223
US 2004-639629P P 20041223
US 2004-639630P P 20041223
US 2004-639631P P 20041223
US 2004-639657P P 20041223
US 2004-639698P P 20041223
US 2005-84624 A2 20050317
US 2005-500240 A2 20050323
US 2005-108088 A2 20050415

AB The present invention relates to use of combinatorial DNA library of mammalian glycosylation enzyme genes for producing modified n-glycans in lower eukaryotes. The invention provides nucleic acid mols. and combinatorial libraries which can be used to successfully target and express mammalian enzymic activities such as those involved in glycosylation to intracellular compartments in a eukaryotic host ***cell*** . Heterologous expression of a set of glycosyltransferases, sugar transporters and mannosidases in eukaryotic host ***cells*** enables oligosaccharide modification and the development of host-strains for the prodn. of mammalian glycoproteins. The process provides an engineered host ***cell*** which can be used to express and target any desirable gene(s) involved in glycosylation. Host ***cells*** with modified oligosaccharides are created or selected. N-glycans made in the engineered host ***cells*** have a Man 5 GlcNAc 2 core structure which may then be modified further by heterologous expression of one or more enzymes, e.g., glycosyltransferases, sugar transporters and mannosidases, to yield human-like glycoproteins. With the primary goal of prodn. of human therapeutic glycoproteins, this method may be adapted to engineer ***cell*** lines in which any desired glycosylation structure may be obtained.

L7 ANSWER 15 OF 36 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:702495 CAPLUS <<LOGINID::20070409>>
DN 141:391680
TI Engineering of an artificial glycosylation pathway blocked in core oligosaccharide assembly in the ***yeast*** Pichia pastoris: Production of complex humanized glycoproteins with terminal galactose
AU Bobrowicz, Piotr; Davidson, Robert C.; Li, Huijuan; Potgieter, Thomas I.; Nett, Juergen H.; Hamilton, Stephen R.; Stadheim, Terrance A.; Miele, Robert G.; Bobrowicz, Beata; Mitchell, Teresa; Rausch, Sebastian; Renfer, Eduard; Wildt, Stefan
CS GlycoFi, Inc., Lebanon, NH, 03766, USA
SO Glycobiology (2004), 14(9), 757-766
CODEN: GLYCE3; ISSN: 0959-6658
PB Oxford University Press
DT Journal
LA English
AB A significant percentage of eukaryotic proteins contain post-translational modifications, including glycosylation, which are required for biol. function. However, the understanding of the structure-function relationships of N-glycans has lagged significantly due to the microheterogeneity of glycosylation in mammalian produced proteins. Recently we reported on the ***cellular*** engineering of ***yeast*** to replicate human N-glycosylation for the prodn. of glycoproteins. Here we report the engineering of an artificial glycosylation pathway in Pichia pastoris blocked in dolichol oligosaccharide assembly. The PpALG3 gene encoding Dol-P-Man:-Man5GlcNAc2-PP-Dol mannosyltransferase was deleted in a strain that was previously engineered to produce hybrid GlcNAcMan5GlcNAc2 human N-glycans. Employing this approach, combined with the use of combinatorial genetic libraries, we engineered P. pastoris strains that synthesize complex GlcNAc2Man3GlcNAc2 N-glycans with striking homogeneity. Furthermore, through expression of a Golgi-localized fusion protein comprising UDP-glucose 4-epimerase and .beta.-1,4-galactosyl transferase activities we demonstrate that this structure is a substrate for highly efficient in vivo galactose addn. Taken together, these data demonstrate that the artificial in vivo glyco-engineering of ***yeast*** represents a major advance in the prodn. of glycoproteins and will emerge as a practical tool to systematically elucidate the structure-function relationship of N-glycans.
RE.CNT 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 16 OF 36 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2004448946 EMBASE <<LOGINID::20070409>>
TI Unaltered complex N-glycan profiles in Nicotiana benthamiana despite drastic reduction of .beta.1,2-N-acetylglucosaminyltransferase I activity.
AU Strasser R.; Altmann F.; Glossl J.; Steinkellner H.
CS Richard.Strasser@boku.ac.at
SO Glycoconjugate Journal, (2004) Vol. 21, No. 5, pp. 275-282. .
Refs: 35
ISSN: 0282-0080 CODEN: GLJOEW
CY Netherlands
DT Journal; Article
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 4 Nov 2004
Last Updated on STN: 4 Nov 2004
AB UDP-GlcNAc:.alpha.3-D-mannoside .beta.1,2-N-acetylglucosaminyltransferase I (GnTI; EC 2.4.1.101) is a Golgi-resident glycosyltransferase that is essential for the processing of oligomannose to hybrid and complex N-glycans in higher eukaryotes. The cDNA of Nicotiana tabacum GnTI has been cloned and characterised previously. To assess the influence of GnTI expression levels on the formation of complex N-glycans we used posttranscriptional gene silencing to knock down the expression of GnTI in the tobacco related species Nicotiana benthamiana. 143 independent transgenic plants containing GnTI constructs in either sense or antisense orientation were generated. 23 lines were selected for measurement of GnTI activity and 10 lines thereof showed a reduction of more than 85% in in vitro assays as compared to wildtype plants. GnTI reduction was stably inherited and did not interfere with the viability of the transformants. Noteworthy one line, 34S/2, exhibited a residual GnTI activity below the detection limit. .beta.1,2-N- ***acetylglucosaminyltransferase*** ***II*** (GnTII), an enzyme which acts further downstream in the N-glycosylation pathway, as well as other control enzymes (.alpha.-mannosidase, .beta.-N-acetylglucosaminidase) were not affected indicating the specific downregulation of GnTI. Remarkably, immunoblots and mass spectrometric N-glycan profiling revealed no significant changes of the total N-glycan pattern. Thus, even the undetectable residual GnTI activity was sufficient for the synthesis of complex N-glycans comparable to wildtype plants.

L7 ANSWER 17 OF 36 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2003:757863 CAPLUS <<LOGINID::20070409>>
DN 139:272048
TI Optimizing protein glycosylation in transgenic plants using ***plant*** /mammalian or mammalian/mammalian (human) chimeric glycosyltransferases for antibody production
IN Bakker, Hendrikus Antonius Cornelus; Florack, Dionisius Elisabeth Antonius; Bosch, Hendrik Jan; Rouwendal, Gerard Johan Adolph
PA Plant Research International B.V., Neth.
SO PCT Int. Appl., 144 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2003078637	A2	20030925	WO 2003-IB1626	20030318
WO 2003078637	A3	20040311		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2478297	A1	20030925	CA 2003-2478297	20030318
AU 2003219418	A1	20030929	AU 2003-219418	20030318
EP 1485486	A2	20041215	EP 2003-715229	20030318
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
JP 2005535291	T	20051124	JP 2003-576629	20030318
CN 1756844	A	20060405	CN 2003-806547	20030318
US 2006253928	A1	20061109	US 2004-508165	20040917

PRAI US 2002-365735P P 20020319

WO 2003-IB1526 W 20030318
WO 2003-IB1626 W 20030318

AB The invention relates to the field of glycoprotein processing in transgenic plants used as cost efficient and contamination safe factories for the prodn. of recombinant glycoproteins and antibodies. The invention is directed to methods for optimizing glycan processing in organisms (and in particular, plants) so that a glycoprotein having complex type bi-antennary N-glycans and thus 5 contg. galactose residues on both arms and which are devoid of (or reduce in) xylose and fucose can be obtained. The invention is further directed to said glycoprotein obtained and host system comprising said protein.

L7 ANSWER 18 OF 36 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2003:551280 CAPLUS <<LOGINID::20070409>>
DN 139:112733
TI Methods for production of recombinant glycoproteins with mammalian-type carbohydrate structures and their use for production of immunoglobulins
IN Wildt, Stefan; Miele, Robert Gordon; Nett, Juergen Hermann; Davidson, Robert C.
PA Glycofi, Inc., USA
SO PCT Int. Appl., 125 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 25

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2003056914	A1	20030717	WO 2002-US41510	20021224
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2471551	A1	20030717	CA 2002-2471551	20021224
AU 2002358296	A1	20030724	AU 2002-358296	20021224
EP 1467615	A1	20041020	EP 2002-792535	20021224
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK				
JP 2005514021	T	20050519	JP 2003-557288	20021224
US 2005170452	A1	20050804	US 2003-500240	20021224
US 2004230042	A1	20041118	US 2003-616082	20030708
US 2005208617	A1	20050922	US 2003-680963	20031007
US 2006040353	A1	20060223	US 2005-108088	20050415
US 2006024292	A1	20060202	US 2005-187065	20050721
US 2006029604	A1	20060209	US 2005-187229	20050721
US 2006034829	A1	20060216	US 2005-187079	20050721
US 2006034830	A1	20060216	US 2005-187113	20050721
US 2006286637	A1	20061221	US 2006-429672	20060505
US 2007037248	A1	20070215	US 2006-546101	20060803
PRAI US 2001-344169P	P	20011227		
US 2000-214358P	P	20000628		
US 2000-215638P	P	20000630		
US 2001-279997P	P	20010330		
US 2001-892591	A2	20010627		
WO 2002-US241510	W	20021224		
WO 2002-US41510	W	20021224		
US 2003-371877	A2	20030220		
US 2003-680963	A	20031007		
WO 2004-US5191	W	20040220		
US 2004-554139P	P	20040317		
US 2004-562424P	P	20040415		
US 2004-589913P	P	20040721		
US 2004-589937P	P	20040721		
US 2004-590011P	P	20040721		
US 2004-590030P	P	20040721		
US 2004-590051P	P	20040721		
US 2004-590052P	P	20040721		
US 2004-639657P	P	20041223		
US 2004-639698P	P	20041223		
US 2005-84624	A2	20050317		
US 2005-500240	A2	20050323		
US 2005-108088	A2	20050415		

AB The present invention relates to host ***cells*** having modified lipid-linked oligosaccharides which may be modified further by heterologous expression of a set of glycosyltransferases, sugar transporters and mannosidases to become host-strains for the prodn. of mammalian, e.g., human therapeutic glycoproteins. The process provides an engineered host ***cell*** which can be used to express and target any desirable gene(s) involved in glycosylation. Host ***cells*** with modified lipid-linked oligosaccharides are created or selected. N-glycans made in the engineered host ***cells*** have a GlcNAcMan3GlcNAc2 core structure which may then be modified further by heterologous expression of one or more enzymes, e.g., glycosyltransferases, sugar transporters and mannosidases, to yield human-like glycoproteins. For the prodn. of therapeutic proteins, this method may be adapted to engineer ***cell*** lines in which any desired glycosylation structure may be obtained. The invention specifically claims use of nucleic acid sequences for gene ALG3 from Pichia pastoris. The ALG3 gene encodes the enzyme which transfers a mannose residue to the Man5-GlcNac2-PP-Dol precursor. The invention also claims use of genetically engineered host ***cells*** for recombinant prodn. of Igs. In examples of the invention, a Pichia pastoris strain with deletions of genes alg3 and och1 was constructed. This strain was transformed with the Kringle 3 domain of human plasminogen as a glycosylation substrate. Mass spectrometric anal. of N-glycans isolated from the kringle 3 glycoproteins showed GlcNAcMan3GlcNAc2 and GlcNAcMan4GlcNAc2 structures which could be further modified in vitro. Addn. of N-acetylglucosamine to GlcNAcMan3GlcNAc2 by N-acetylglucosaminyltransferases II and III yields a "bisected" N-glycan, GlcNAc3Man3GlcNAc2, which has been implicated in greater antibody-dependent ***cellular*** cytotoxicity. Methods of the invention can be used to engineer a ***yeast*** strain capable of producing glycoproteins with bisected N-glycans and expressing Ig mols. with bisected N-glycans attached to asparagine residue 297 in the CH2 portion.
RE.CNT 15 THERE ARE 15 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 19 OF 36 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2003:464395 CAPLUS <<LOGINID::20070409>>
DN 140:250298
TI Two closely related forms of UDP-GlcNAc:.alpha.6-D-mannoside .beta.1,2-N-***acetylglucosaminyltransferase*** ***II*** occur in the clawed frog Xenopus laevis
AU Mucha, Jan; Svoboda, Barbara; Kappel, Sonja; Strasser, Richard; Bencur, Peter; Froehwein, Ulrike; Schachter, Harry; Mach, Lukas; Gloessl, Josef
CS Zentrum fuer Angewandte Genetik, Universitaet fuer Bodenkultur Wien, Vienna, A-1190, Austria
SO Glycoconjugate Journal (2003), Volume Date 2002, 19(3), 187-195
CODEN: GLJOEW; ISSN: 0282-0080
PB Kluwer Academic Publishers
DT Journal
LA English
AB UDP-GlcNAc:.alpha.6-D-mannoside .beta.1,2-N-***acetylglucosaminyltransferase*** ***II*** (GnT ***II*** ; EC 2.4.1.143) is a medial-Golgi resident enzyme that catalyzes an essential step in the biosynthetic pathway leading from high mannose to complex N-linked oligosaccharides. Screening a cDNA library from X. laevis ovary with a human GnT II DNA probe resulted in the isolation of 2 cDNA clones encoding 2 closely related GnT II isoenzymes, GnT II-A and GnT II-B. Anal. of the corresponding genomic DNAs revealed that the open reading frame of both X. laevis GnT II genes resides within a single exon. The GnT II-A gene was found to be transcriptionally active in all X. laevis tissues tested. In contrast, expression of the GnT II-B gene was detected only in a limited no. of tissues. Both GnT II-A and GnT II-B exhibit a type II transmembrane protein topol. with a putative N-terminal cytoplasmic tail of 9 amino acids followed by a transmembrane domain of 18 residues, and a C-terminal luminal domain of 405 residues. The 2 proteins differ at 28 amino acid positions within their luminal regions. Heterologous expression of sol. forms of the enzymes in ***insect*** ***cells*** showed that GnT II-A and GnT II-B are both catalytically active and exhibit similar specific activities. Both recombinant proteins are modified with N-linked oligosaccharides. N-terminal deletion studies demonstrated that the 1st 49 amino acid residues are not essential for proper folding and enzymic activity of X. laevis GnT II.
RE.CNT 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 20 OF 36 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN DUPLICATE 3
AN 2003135027 EMBASE <<LOGINID::20070409>>
TI Complex-type biantennary N-glycans of recombinant human transferrin from Trichoplusia ni ***insect*** ***cells*** expressing mammalian .beta.-1,4-galactosyltransferase and .beta.-1,2-N-***acetylglucosaminyltransferase*** ***II*** .
AU Tomiya N.; Howe D.; Aumiller J.J.; Pathak M.; Park J.; Palter K.B.; Jarvis D.L.; Betenbaugh M.J.; Lee Y.C.
CS N. Tomiya, Department of Biology, Johns Hopkins University, 3400 North Charles Street, Baltimore, MD 21218, United States. ntomiya1@jhu.edu
SO Glycobiology, (1 Jan 2003) Vol. 13, No. 1, pp. 23-34. .
Refs: 47
ISSN: 0959-6658 CODEN: GLYCE3
CY United Kingdom
DT Journal; Article
FS 004 Microbiology
LA English
SL English
ED Entered STN: 17 Apr 2003
Last Updated on STN: 17 Apr 2003
AB A novel recombinant baculovirus expression vector was used to produce His-tagged human transferrin in a transformed ***insect*** ***cell*** line (Tn5.beta.4GalT) that constitutively expresses a mammalian .beta.-1,4-galactosyltransferase. This virus encoded the His-tagged human transferrin protein in conventional fashion under the control of the very late polyhedrin promoter. In addition, to enhance the synthesis of galactosylated biantennary N-glycans, this virus encoded human .beta.-1,2-N- ***acetylglucosaminyltransferase*** ***II*** under the control of an immediate-early (ie1) promoter. Detailed analyses by MALDI-TOF MS, exoglycosidase digestion, and two-dimensional HPLC revealed that the N-glycans on the purified recombinant human transferrin

produced by this virus-host system included four different fully galactosylated, biantennary, complex-type glycans. Thus, this study describes a novel baculovirus-host system, which can be used to produce a recombinant glycoprotein with fully galactosylated, biantennary N-glycans.

L7 ANSWER 21 OF 36 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN DUPLICATE 4
AN 2002458926 EMBASE <<LOGINID::20070409>>
TI Engineering the protein N-glycosylation pathway in ***insect*** ***cells*** for production of biantennary, complex N-glycans.
AU Hollister J.; Grabenhorst E.; Nimtz M.; Conradt H.; Jarvis D.L.
CS D.L. Jarvis, Department of Molecular Biology, University of Wyoming, Laramie, WY 82071, United States. dljarvis@uwyo.edu
SO Biochemistry, (17 Dec 2002) Vol. 41, No. 50, pp. 15093-15104. .
Refs: 47
ISSN: 0006-2960 CODEN: BICHAW
CY United States
DT Journal; Article
FS 004 Microbiology
LA English
SL English
ED Entered STN: 9 Jan 2003
Last Updated on STN: 9 Jan 2003
AB ***Insect*** ***cells*** , like other eucaryotic ***cells*** , modify many of their proteins by N-glycosylation. However, the endogenous ***insect*** ***cell*** N-glycan processing machinery generally does not produce complex, terminally sialylated N-glycans such as those found in mammalian systems. This difference in the N-glycan processing pathways of ***insect*** ***cells*** and higher eucaryotes imposes a significant limitation on their use as hosts for baculovirus-mediated recombinant glycoprotein production. To address this problem, we previously isolated two transgenic ***insect*** ***cell*** lines that have mammalian .beta.1,4-galactosyltransferase or .beta.1,4-galactosyltransferase and .alpha.2,6-sialyltransferase genes. Unlike the parental ***insect*** ***cell*** line, both transgenic ***cell*** lines expressed the mammalian glycosyltransferases and were able to produce terminally galactosylated or sialylated N-glycans. The purpose of the present study was to investigate the structures of the N-glycans produced by these transgenic ***insect*** ***cell*** lines in further detail. Direct structural analyses revealed that the most extensively processed N-glycans produced by the transgenic ***insect*** ***cell*** lines were novel, monoantennary structures with elongation of only the .alpha.1,3 branch. This led to the hypothesis that the transgenic ***insect*** ***cell*** lines lacked adequate endogenous N- ***acetylglucosaminyltransferase*** ***II*** activity for biantennary N-glycan production. To test this hypothesis and further extend the N-glycan processing pathway in Sf9 ***cells*** , we produced a new transgenic line designed to constitutively express a more complete array of mammalian glycosyltransferases, including N- ***acetylglucosaminyltransferase*** ***II*** . This new transgenic ***insect*** ***cell*** line, designated SfSWT-1, has higher levels of five glycosyltransferase activities than the parental ***cells*** and supports baculovirus replication at normal levels. In addition, direct structural analyses showed that SfSWT-1 ***cells*** could produce biantennary, terminally sialylated N-glycans. Thus, this study provides new insight on the glycobiology of ***insect*** ***cells*** and describes a new transgenic ***insect*** ***cell*** line that will be widely useful for the production of more authentic recombinant glycoproteins by baculovirus expression vectors.

L7 ANSWER 22 OF 36 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2003288080 EMBASE <<LOGINID::20070409>>
TI Two closely related forms of UDP-GlcNAc: .alpha.6-D-mannoside .beta.1,2-N-acetylglucosaminyl-transferase II occur in the clawed frog Xenopus laevis.
AU Mucha J.; Svoboda B.; Kappel S.; Strasser R.; Bencur P.; Frohwein U.; Schachter H.; Mach L.; Glossl J.
CS L. Mach, Zentrum fur Angewandte Genetik, Univ. fur Bodenkultur Wien, Muthgasse 18, A-1190 Wien, Austria. lukas.mach@boku.ac.at
SO Glycoconjugate Journal, (1 Mar 2003) Vol. 19, No. 3, pp. 187-195. .
Refs: 31
ISSN: 0282-0080 CODEN: GLJOEW
CY Netherlands
DT Journal; Article
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 31 Jul 2003
Last Updated on STN: 31 Jul 2003
AB UDP-GlcNAc:.alpha.6-D-mannoside .beta.1,2-N- ***acetylglucosaminyltransferase*** ***II*** (GnT ***II*** ; EC 2.4.1.143) is a medial-Golgi resident enzyme that catalyses an essential step in the biosynthetic pathway leading from high mannose to complex N-linked oligosaccharides. Screening a cDNA library from Xenopus laevis ovary with a human GnT II DNA probe resulted in the isolation of two cDNA clones encoding two closely related GnT II isoenzymes, GnT II-A and GnT II-B. Analysis of the corresponding genomic DNAs revealed that the open reading frame of both X. laevis GnT II genes resides within a single exon. The GnT II-A gene was found to be transcriptionally active in all X. laevis tissues tested. In contrast, expression of the GnT II-B gene was detected only in a limited number of tissues. Both GnT II-A and GnT II-B exhibit a type II transmembrane protein topology with a putative N-terminal cytoplasmic tail of 9 amino acids followed by a transmembrane domain of 18 residues, and a C-terminal luminal domain of 405 residues. The two proteins differ at 28 amino acid positions within their luminal regions. Heterologous expression of soluble forms of the enzymes in ***insect*** ***cells*** showed that GnT II-A and GnT II-B are both catalytically active and exhibit similar specific activities. Both recombinant proteins are modified with N-linked oligosaccharides. N-terminal deletion studies demonstrated that the first 49 amino acid residues are not essential for proper folding and enzymatic activity of X. laevis GnT II.

L7 ANSWER 23 OF 36 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2001:557217 BIOSIS <<LOGINID::20070409>>
DN PREV200100557217
TI Structural analyses of N-glycans produced by novel transgenic ***insect*** ***cell*** lines.
AU Hollister, Jason R. [Reprint author]; Grabenhorst, Eckart; Nimtz, Manfred; Conradt, Harald S.; Jarvis, Donald L. [Reprint author]
CS Department of Molecular Biology, University of Wyoming, Laramie, Wyoming, 82071, USA
SO Glycobiology, (October, 2001) Vol. 11, No. 10, pp. 925. print.
Meeting Info.: 6th Annual Conference of the Society for Glycobiology. San Francisco, California, USA. November 14-17, 2001.
ISSN: 0959-6658.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 5 Dec 2001
Last Updated on STN: 25 Feb 2002

L7 ANSWER 24 OF 36 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2001335998 EMBASE <<LOGINID::20070409>>
TI Congenital disorders of glycosylation type Ia and IIa are associated with different primary haemostatic complications.
AU Van Geet C.; Jaeken J.; Freson K.; Lenaerts T.; Arnout J.; Vermylen J.; Hoylaerts M.F.
CS C. Van Geet, Department of Paediatrics, UZ Gasthuisberg, University of Leuven, Herestraat 49, 3000 Leuven, Belgium. Christel.Vangeet@uz.kuleuven.ac.be
SO Journal of Inherited Metabolic Disease, (2001) Vol. 24, No. 4, pp. 477-492. .
Refs: 18
ISSN: 0141-8955 CODEN: JIMDDP
CY Netherlands
DT Journal; Article
FS 025 Hematology
029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 11 Oct 2001
Last Updated on STN: 11 Oct 2001
AB Congenital disorders of glycosylation (CDG) type I are mostly due to a deficient phosphomannomutase activity, called CDG Ia. CDG IIa (mutations in the MGAT2 gene) results from a deficient activity of the Golgi enzyme N- ***acetylglucosaminyltransferase*** ***II*** . CDG Ia patients predominantly have a thrombotic tendency, whereas our CDG IIa patient has an increased bleeding tendency, despite similar coagulation factor abnormalities in both types. We have investigated whether abnormally glycosylated platelet membrane glycoproteins are involved in the haemostatic complications of both CDG groups. In flow cytometry, the binding of Ricinus communis lectin (reactive with .beta.-galactose primarily) to control platelets increased after neuraminidase treatment: this increase was smaller ($p < 0.01$) in CDG Ia patients (3.1 .+-. 0.08 times) than in control platelets (8.5 .+-. 1.8 times) and did not occur in the CDG IIa patient. Platelet-rich plasma from CDG Ia patients, but not a CDG IIa patient, aggregated spontaneously and gel-filtered platelets from CDG Ia patients agglutinated at very low concentrations of ristocetin, independently of von Willebrand factor (vWF). Accordingly, in stirred whole blood, the rate of single platelet disappearance of CDG Ia patients was twice that of control platelets. In contrast, perfusion of whole anticoagulated blood of the CDG IIa patient over collagen yielded markedly decreased platelet adherence to collagen at shear rates involving glycoprotein (GP) Ib-vWF interactions. Thus, abnormal glycosylation of platelet glycoproteins in CDG Ia enhances nonspecific platelet interactions, in agreement with a thrombotic tendency. The reduced GP Ib-mediated platelet reactivity with vessel wall components in the CDG IIa patient under flow conditions provides a basis for his bleeding tendency.

L7 ANSWER 25 OF 36 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN DUPLICATE 5
AN 2001094002 EMBASE <<LOGINID::20070409>>
TI A new .beta.-1,2-N-acetylglucosaminyltransferase that may play a role in the biosynthesis of mammalian O-mannosyl glycans.
AU Takahashi S.; Sasaki T.; Manya H.; Chiba Y.; Yoshida A.; Mizuno M.; Ishida H.-K.; Ito F.; Inazu T.; Kotani N.; Takasaki S.; Takeuchi M.; Endo T.
CS T. Endo, Department of Glycobiology, Tokyo Metropolitan Inst. Gerontology, 35-2 Sakaecho, Itabashi-ku, Tokyo 173-0015, Japan
SO Glycobiology, (2001) Vol. 11, No. 1, pp. 37-45. .
Refs: 41
ISSN: 0959-6658 CODEN: GLYCE3

CY United Kingdom
DT Journal; Article
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 29 Mar 2001
Last Updated on STN: 29 Mar 2001
AB Recent studies have shown that O-mannosyl glycans are present in several mammalian glycoproteins. Although knowledge on the functional roles of these glycans is accumulating, their biosynthetic pathways are poorly understood. Here we report the identification and initial characterization of a novel enzyme capable of forming GlcNAc.beta.1-2Man linkage, namely UDP-N-acetyl-glucosamine: O-linked mannose .beta.-1,2-N-acetylglucosaminyl-transferase in the microsome fraction of newborn rat brains. The enzyme transfers GlcNAc to .beta.-linked mannose residues, and the formed linkage was confirmed to be .beta.1-2 on the basis of diplococcal .beta.-N-acetylhexosaminidase susceptibility and by high-pH anion-exchange chromatography. Its activity is linearly dependent on time, protein concentration, and substrate concentration and is enhanced in the presence of manganese ion. Its activity is not due to UDP-N-acetylglucosamine: .alpha.-3-D-mannoside .beta.-1,2-N-acetylglucosaminyl- transferase I (GnT-I) or UDP-N-acetylglucosamine: .alpha.-6-D-mannoside .beta.-1,2-D- ***acetylglucosaminyltransferase*** ***II*** (GnT- ***II***), which acts on the early steps of N-glycan biosynthesis, because GnT-I or GnT-II expressed in ***yeast*** ***cells*** did not show any GlcNAc transfer activity against a synthetic mannosyl peptide. Taken together, the results suggest that the GlcNAc transferase activity described here is relevant to the O-mannosyl glycan pathway in mammals.

L7 ANSWER 26 OF 36 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2001:8126 CAPLUS <<LOGINID::20070409>>
DN 134:233435
TI Molecular cloning of cDNA encoding N- ***acetylglucosaminyltransferase*** ***II*** from Arabidopsis thaliana
AU Strasser, R.; Steinkellner, H.; Boren, M.; Altmann, F.; Mach, L.; Glossl, J.; Mucha, J.
CS Zentrum fur Angewandte Genetik, Universitat fur Bodenkultur Wien, Vienna, 1190, Austria
SO Glycoconjugate Journal (2000), Volume Date 1999, 16(12), 787-791
CODEN: GLJOEW; ISSN: 0282-0080
PB Kluwer Academic Publishers
DT Journal
LA English
AB N- ***acetylglucosaminyltransferase*** ***II*** (GnTII, E.C. 2.4.1.143) is a Golgi enzyme involved in the biosynthesis of glycoprotein-bound N-linked oligosaccharides, catalyzing an essential step in the conversion of oligomannose-type to complex N-glycans. GnTII activity has been detected in both animals and plants. However, while cDNAs encoding the enzyme have already been cloned from several mammalian
sources no GnTII homolog has been cloned from plants so far. Here we report the mol. cloning of an Arabidopsis thaliana GnTII cDNA with striking homol. to its animal counterparts. The predicted domain structure of A. thaliana GnTII indicates a type II transmembrane protein topol. as it has been established for the mammalian variants of the enzyme. Upon expression of A. thaliana GnTII cDNA in the baculovirus/ ***insect*** ***cell*** system, a recombinant protein was produced that exhibited GnTII activity.
RE.CNT 17 THERE ARE 17 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 27 OF 36 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on
STN
AN 2001:37545 BIOSIS <<LOGINID::20070409>>
DN PREV200100037545
TI Kinetic basis for the donor nucleotide-sugar specificity of beta1,4-N- ***acetylglucosaminyltransferase*** ***III*** .
AU Ikeda, Yoshitaka; Koyota, Souichi; Ihara, Hideyuki; Yamaguchi, Yukihiro; Korekane, Hiroaki; Tsuda, Takeo; Sasai, Ken; Taniguchi, Naoyuki [Reprint author]
CS Department of Biochemistry, Osaka University Medical School, 2-2 Yamadaoka, Suita, Osaka, 565-0871, Japan
proftani@biochem.med.osaka-u.ac.jp
SO Journal of Biochemistry (Tokyo), (Oct., 2000) Vol. 128, No. 4, pp. 609-619. print.
CODEN: JOBIAO. ISSN: 0021-924X.
DT Article
LA English
ED Entered STN: 17 Jan 2001
Last Updated on STN: 12 Feb 2002
AB The kinetic basis of the donor substrate specificity of beta1,4-N- ***acetylglucosaminyltransferase*** ***III*** (GnT- ***III***) was investigated using a purified recombinant enzyme. The enzyme also transfers GalNAc and Glc moieties from their respective UDP-sugars to an acceptor at rates of 0.1-0.2% of that for GlcNAc, but Gal is not transferred at a detectable rate. Kinetic analyses revealed that these inefficient transfers, which are associated with the specificity of the enzyme, are due to the much lower Vmax values, whereas the Km values for UDP-GalNAc and UDP-Glc differ only slightly from that for UDP-GlcNAc. It was also found that various other nucleotide-Glc derivatives bind to the enzyme with comparable affinities to those of UDP-GlcNAc and UDP-Glc, although the derivatives do not serve as glycosyl donors. Thus, GnT-III does not appear to distinguish UDP-GlcNAc from other structurally similar nucleotide-sugars by specific binding in the ground state. These findings suggest that the specificity of GnT-III toward the nucleotide-sugar is determined during the catalytic process. This type of specificity may be efficient in preventing a possible mistransfer when other nucleotide-sugars are present in excess over the true donor.

L7 ANSWER 28 OF 36 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN DUPLICATE 6
AN 2000427506 EMBASE <<LOGINID::20070409>>
TI Molecular cloning of cDNA encoding N- ***acetylglucosaminyltransferase*** ***II*** from Arabidopsis thaliana.
AU Strasser R.; Steinkellner H.; Boren M.; Altmann F.; Mach L.; Glossl J.; Mucha J.
CS H. Steinkellner, Zentrum for Angewandte Genetik, Universitat fur Bodenkultur Wien, Muthgasse 18, 1190 Wien, Austria. steink@mail.boku.ac.at
SO Glycoconjugate Journal, (1999) Vol. 16, No. 12, pp. 787-791. .
Refs: 18
ISSN: 0282-0080 CODEN: GLJOEW
CY Netherlands
DT Journal; Article
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 21 Dec 2000
Last Updated on STN: 21 Dec 2000
AB N- ***acetylglucosaminyltransferase*** ***II*** (GnTII, EC 2.4.1.143) is a Golgi enzyme involved in the biosynthesis of glycoproteinbound N-linked oligosaccharides, cetalysing an essential step in the conversion of oligomannose-type to complex Nglycans. GnTII activity has been detected in both animals and plants. However, while cDNAs encoding the enzyme have already been cloned from several mammalian
sources no GnTII homologue has been cloned from plants so far. Here we report the molecular cloning of an Arabidopsis thaliana GnTII cDNA with striking homology to its animal counterparts. The predicted domain structure of A. thaliana GnTII indicates a type II transmembrane protein topology as it has been established for the mammalian variants of the enzyme. Upon expression of A. thaliana GnTII cDNA in the baculovirus/ ***insect*** ***cell*** system, a recombinant protein was produced that exhibited GnTII activity.

L7 ANSWER 29 OF 36 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1999:470715 CAPLUS <<LOGINID::20070409>>
DN 131:238578
TI Cloning and the expression of the murine gene and chromosomal location of the human gene encoding N-acetylglucosaminyltransferase I. [Erratum to document cited in CA119:174988]
AU Kumar, Ravindra; Yang, Jing; Eddy, Roger L.; Byers, Mary G.; Shows, Thomas
B.; Stanley, Pamela
CS Dep. Cell Biol., Albert Einstein College Medicine, New York, NY, 10461, USA
SO Glycobiology (1999), 9(8), ix
CODEN: GLYCE3; ISSN: 0959-6658
PB Oxford University Press
DT Journal
LA English
AB In the article, MGAT1 was mapped to human 5q31.2-q31.3. However, Tan et al. ("The human UDP-N-acetylglucosamine: .alpha.-6-D-mannoside-.beta.-1,2-N- ***acetylglucosaminyltransferase*** ***II*** gene (MGAT2). Cloning of genomic DNA, localization to chromosome 14q21, expression in ***insect*** ***cells*** and purifn. of the recombinant protein," Eur. J. Biochem., 231, 317-328, 1995) subsequently mapped MGAT1 to 5q35 using FISH. Using more sensitive FISH technol. than in the 1992 report, MGAT1 was found to indeed be located at 5135. The original 1.3 kb genomic probe, a new 2.8 kb cDNA probe, a new 5 kb genomic probe, and FISH technol. were employed on human leukocytes to confirm the 5q35 location. Double signals were seen on both chromatids at 5935 using all three probes.

L7 ANSWER 30 OF 36 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN DUPLICATE 7
AN 96301016 EMBASE <<LOGINID::20070409>>
DN 1996301016
TI Mutations in the MGAT2 gene controlling complex N-glycan synthesis cause carbohydrate-deficient glycoprotein syndrome type II, an autosomal recessive disease with defective brain development.
AU Tan J.; Dunn J.; Jaeken J.; Schachter H.
CS Department of Biochemistry Research, Hospital for Sick Children, 555 University Avenue,Toronto, Ont. M5G 1X8, Canada
SO American Journal of Human Genetics, (1996) Vol. 59, No. 4, pp. 810-817. .
ISSN: 0002-9297 CODEN: AJHGAG
CY United States
DT Journal; Article
FS 022 Human Genetics
029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 28 Oct 1996
Last Updated on STN: 28 Oct 1996

AB Carbohydrate-deficient glycoprotein syndrome (CDGS) type II is a multisystemic congenital disease with severe involvement of the nervous system. Two unrelated CDGS type II patients are shown to have point mutations (one patient having Ser.fwdarw.Phe and the other having His.fwdarw.Arg) in the catalytic domain of the gene MGAT2, encoding UDP-GlcNAc:.alpha.-6-D-mannoside .beta.-1,2-N- ***acetylglucosaminyltransferase*** ***II*** (GnT ***II***), an enzyme essential for biosynthesis of complex Asn-linked glycans. Both mutations caused both decreased expression of enzyme protein in a baculovirus/ ***insect*** ***cell*** system and inactivation of enzyme activity. Restriction-endonuclease analysis of DNA from 23 blood relatives of one of these patients showed that 13 donors were heterozygotes; the other relatives and 21 unrelated donors were normal homozygotes. All heterozygotes showed a significant reduction (33%-68%) in mononuclear- ***cell*** GnT II activity. The data indicate that CDGS type II is an autosomal recessive disease and that complex Asn-linked glycans are essential for normal neurological development.

L7 ANSWER 31 OF 36 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN DUPLICATE 8
AN 95225205 EMBASE <<LOGINID::20070409>>
DN 1995225205
TI The human UDP-N-acetylglucosamine: .alpha.-6-D-mannoside-.beta.-1,2-N- ***acetylglucosaminyltransferase*** ***II*** gene (MGAT2) - Cloning of genomic DNA, localization to chromosome 14q21, expression in ***insect*** ***cells*** and purification of the recombinant protein.
AU Tan J.; D'Agostaro G.A.F.; Bendiak B.; Reck F.; Sarkar M.; Squire J.A.; Leong P.; Schachter H.
CS Department of Biochemistry, Hospital for Sick Children, 555 University Avenue,Toronto, Ont. M5G 1X8, Canada
SO European Journal of Biochemistry, (1995) Vol. 231, No. 2, pp. 317-328. . ISSN: 0014-2956 CODEN: EJBCAI
CY Germany
DT Journal; Article
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 22 Aug 1995
Last Updated on STN: 22 Aug 1995
AB UDP-GlcNAc:.alpha.-6-D-mannoside [GlcNAc to Man.alpha.1-6] .beta.-1,2-N- ***acetylglucosaminyltransferase*** ***II*** (GlcNAc-T ***II*** , EC 2.4.1.143) is a Golgi enzyme catalyzing an essential step in the conversion of oligo-mannose to complex N-glycans. A 12-kb probe from a rat liver cDNA encoding GlcNAc-T II was used to screen a human genomic DNA library in .lambda.EMBL3. Southern analysis of restriction endonuclease digests of positive phage clones identified two hybridizing fragments (3.0 and 3.5 kb) which were subcloned into pBlueScript. The inserts of the resulting plasmids (pHG30 and pHG36) are over-lapping clones containing 5.5 kb of genomic DNA. The pHG30 insert (3.0 kb) contains a 1341-bp open reading frame encoding a 447-amino-acid protein, 250 bp of G+C- rich 5'-upstream sequence and 1.4 kb of 3' downstream sequence. The pHG36 insert (3.5 kb) contains 2.75 kb of 5'-upstream sequence and 750 bp of the 5'-end of the open reading frame. The protein sequence showed the domain structure typical of all previously cloned glycosyltransferases, i.e. a short 9-residue putative cytoplasmic N-terminal domain, a 20-residue hydrophobic non-cleavable putative signal-anchor domain and a 418-residue C-terminal catalytic domain Northern analysis of human tissues showed a major message at 3 kb and minor signals at 2 and 4.5 kb. There is no sequence similarity to any previously cloned glycosyltransferases including human UDP-GlcNAc:.alpha.-3- D-mannoside [GlcNAc to Man.alpha.1-3] .beta.-1,2-N-acetylglucosaminyltransferase I (GlcNAc-T I) which has 445 amino acids with a 418-residue C-terminal catalytic domain. The human GlcNAc-T I and II genes (MGAT1 and MGAT2) map to chromosome bands 5q35 and 14q21, respectively, by fluorescence in situ hybridization. The entire coding regions of human GlcNAc-T I and II are each on a single exon. There is 92% identity between the amino acid sequences of the catalytic domains of human and rat GlcNAc-T II. Southern analysis of restriction enzyme digests of human genomic DNA indicates that there is only a single copy of the MGAT2 gene. The full-length coding region of GlcNAc-T II has been expressed in the baculovirus/Sf9 ***insect*** ***cell*** system, the recombinant enzyme has been purified to near homogeneity with a specific activity of about 20 .mu.mol .cntdot. min-1 .cntdot. mg-1 and the product synthesized by the recombinant enzyme has been identified by high-resolution 1H-NMR spectroscopy and mass spectrometry.

L7 ANSWER 32 OF 36 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN DUPLICATE 9
AN 95302668 EMBASE <<LOGINID::20070409>>
DN 1995302668
TI Synthesis of pentasaccharide analogues of the N-glycan substrates of N-acetylglucosaminyltransferases III, IV and V using tetrasaccharide precursors and recombinant .beta.-(1 .fwdarw. 2)-N- ***acetylglucosaminyltransferase*** ***II*** .
AU Reck F.; Meinjohanns E.; Tan J.; Grey A.A.; Paulsen H.; Schachter H.
CS Research Institute, Hospital for Sick Children,Toronto, Ont. M5G 1X8, Canada
SO Carbohydrate Research, (1995) Vol. 275, No. 2, pp. 221-229. . ISSN: 0008-6215 CODEN: CRBRAT
CY Netherlands
DT Journal; Article
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 11 Nov 1995
Last Updated on STN: 11 Nov 1995
AB Recombinant human UDP-GlcNAc: .alpha.-Man-(1 .fwdarw. 6)R .beta.-(1 .fwdarw. 2)-N- ***acetylglucosaminyltransferase*** ***II*** (EC 2.4.1.143, GlcNAc-T II) was produced in the Sf9 ***insect*** ***cell*** /baculovirus expression system as a fusion protein with a (His)6 tag and partially purified by affinity chromatography on a metal chelating column. The partially purified enzyme was used to catalyze the transfer of GlcNAc from UDP-GlcNAc to R-.alpha.-Man(1 .fwdarw. 6)(.beta.-GlcNAc(1 .fwdarw. 2).alpha.-Man(1 .fwdarw. 3)).beta.-Man-O-octyl to form .beta.-GlcNAc(1 .fwdarw. 2)R-.alpha.-Man(1 .fwdarw. 6)(.beta.-GlcNAc(1 .fwdarw. 2).alpha.-Man(1 .fwdarw. 3)).beta.-Man-O-octyl where there is either no modification of the .alpha.-Man(1 .fwdarw. 6) residue (7), or where R is 3-deoxy (8), 4-deoxy (9) or 6-deoxy (10). The yields ranged from 64-80%. Products were characterized by 1H and 13C nuclear magnetic resonance spectroscopy and fast atom bombardment mass spectrometry. Compounds 7-10 are pentasaccharide analogues of the biantennary N-glycan substrates of N-acetylglucosaminyltransferases III, IV and V.

L7 ANSWER 33 OF 36 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN DUPLICATE 10
AN 94159450 EMBASE <<LOGINID::20070409>>
DN 1994159450
TI Synthesis of tetrasaccharide analogues of the N-glycan substrate of .beta.-(1 .fwdarw. 2)-N- ***acetylglucosaminyltransferase*** ***II*** using trisaccharide precursors and recombinant .beta.-(1 .fwdarw. 2)-N-acetylglucosaminyltransferase I.
AU Reck F.; Springer M.; Paulsen H.; Brockhausen I.; Sarkar M.; Schachter H.
CS Research Institute, Hospital for Sick Children,Toronto, Ont. M5G 1X8, Canada
SO Carbohydrate Research, (1994) Vol. 259, No. 1, pp. 93-101. . ISSN: 0008-6215 CODEN: CRBRAT
CY Netherlands
DT Journal; Article
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 22 Jun 1994
Last Updated on STN: 22 Jun 1994
AB Recombinant rabbit UDP-GlcNAc : .alpha.-Man-(1 .fwdarw. 3R) .beta.-(1 .fwdarw. 2)-N-acetylglucosaminyltransferase I (EC 2.4.1.101, GlcNAc-T I) produced in the Sf9 ***insect*** ***cell*** /baculovirus expression system has been used to convert compounds of the form 3-R-.alpha.-Man(1 .fwdarw. 6)(.alpha.-Man(1 .fwdarw. 3)).beta.-Man-O-octyl to 3-R-.alpha.-Man(1 .fwdarw. 6)(.beta.-GlcNAc(1 .fwdarw. 2).alpha.-Man(1 .fwdarw. 3)).beta.-Man-O-octyl where R is OH (14), O-methyl (17), O-pentyl (18), O-(4,4-azo)pentyl (19), O-(5-iodoacetamido)pentyl (20) and O-(5-amino)pentyl (21); 2-deoxy-.alpha.-Man(1 .fwdarw. 6)(.beta.-GlcNAc(1 .fwdarw. 2).alpha.-Man(1 .fwdarw. 3)).beta.-Man-O-octyl (16), 4-O-methyl-.alpha.-Man(1 .fwdarw. 6)(.beta.-GlcNAc(1 .fwdarw. 2).alpha.-Man(1 .fwdarw. 3)).beta.-Man-O-octyl (22), 6-O-methyl-.alpha.-Man(1 .fwdarw. 6)(.beta.-GlcNAc(1 .fwdarw. 2).alpha. Man(1 .fwdarw. 3)).beta.-Man-O-octyl (23) and .alpha.-Man(1 .fwdarw. 6)[.beta.-GlcNAc(1 .fwdarw. 2)(4-O-methyl).alpha.-Man(1 .fwdarw. 3)].beta.-Man-O-octyl (15) were also synthesized by this procedure. The yields ranged from 80 to 99%. Products were characterized by high resolution 1H and 13C nuclear magnetic resonance spectroscopy and fast atom bombardment mass spectrometry. Compounds 14, 15, 17, 22, and 23 are excellent substrates for UDP-GlcNAc: .alpha.-Man(1 .fwdarw. 6R) .beta.-(1 .fwdarw. 2)-N- ***acetylglucosaminyltransferase*** ***II*** and the other compounds are inhibitors of this enzyme.

L7 ANSWER 34 OF 36 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN DUPLICATE 11
AN 94027189 EMBASE <<LOGINID::20070409>>
DN 1994027189
TI Processing of asparagine-linked oligosaccharides in ***insect*** ***cells*** . N- ***acetylglucosaminyltransferase*** I and ***II*** activities in cultured lepidopteran ***cells*** .
AU Altmann F.; Kornfeld G.; Dalik T.; Staudacher E.; Glossl J.
CS Institut fur Chemie, Universitat fur Bodenkultur, Gregor-Mendelstrasse 33,A-1180 Wien, Austria
SO Glycobiology, (1993) Vol. 3, No. 6, pp. 619-625. . ISSN: 0959-6658 CODEN: GLYCE3
CY United Kingdom
DT Journal; Article
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 20 Feb 1994
Last Updated on STN: 20 Feb 1994
AB The levels of .beta.1,2-N-acetylglucosaminyltransferase (GlcNAc-T) I and II activities in cultured ***cells*** from Bombyx mori (Bm-N), Mamestra brassicae (IZD-Mb-0503) and Spodoptera frugiperda (Sf-9 and Sf-21) were investigated. Apart from initial experiments with Man.alpha.-3(Man.alpha.1-6)Man.beta.1-O(CH2)8COOH3 and 3H-labelled UDP-GlcNAc as substrates, GlcNAc-T I activity was measured with a nonradioactive HPLC method using pyridylaminated Man3GlcNAc2 and Man5GlcNAc2 as acceptor oligosaccharides. It was shown by reversed-phase HPLC, exoglycosidase digestion and methylation analysis that the product

obtained with Man3GlcNAc2 contained a terminal GlcNAc residue linked .beta.1,2 to the .alpha.1,3 arm of the acceptor. Compared to the enzyme from the human hepatoma ***cell*** line HepG2, ***insect*** ***cell*** GlcNAc-T I exhibited a much higher preference for the Man5 substrate. The GlcNAc-T I from Mb-0503 ***cells*** had apparent K(m) and V(max) values for pyridylaminated Man3- and Man5GlcNAc2 of 2.15 and 0.21 mM, and of 3.4 and 11.4 nmol/h/mg of ***cell*** protein, respectively. When Man5GlcNAc2 was used as the acceptor substrate, the levels of GlcNAc-T I activity in the four ***insect*** ***cell*** lines ranged between 7.5 and 14.7 nmol/h/mg of ***cell*** protein, and thus were comparable to that of HepG2 ***cells*** . Evidence is presented for the dependence of lepidopteran fucosyltransferase on the presence of terminal N-acetylglucosamine. GlcNAc-T II activity could be demonstrated by HPLC using GlcNAc.beta.1-2Man.alpha.1-3(Man.alpha.1-6)Man.beta.1-4GlcNAc.beta.1-4GlcNAc-pyridylamine as the acceptor in the presence of 6-acetamido-6-deoxycastanospermine as an inhibitor of .beta.-N-acetylglucosaminidase. However, the ***insect*** ***cells*** exhibited specific activities of GlcNAc-T II of only 0.02-0.11 nmol/h/mg of ***cell*** protein, much less than HepG2 ***cells*** .

L7 ANSWER 35 OF 36 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1992:230609 CAPLUS <<LOGINID::20070409>>
DN 116:230609
TI Studies on synthetic pathway of xylose-containing N-linked oligosaccharides deduced from substrate specificities of the processing enzymes in sycamore ***cells*** (Acer pseudoplatanus L.)
AU Tezuka, Katsunari; Hayashi, Makoto; Ishihara, Hideko; Akazawa, Takashi; Takahashi, Noriko
CS Fac. Pharm. Sci., Nagoya City Univ., Nagoya, 467, Japan
SO European Journal of Biochemistry (1992), 203(3), 401-13
CODEN: EJBCAI; ISSN: 0014-2956
DT Journal
LA English
AB The activities of .alpha.-1,3-mannosyl-glycoprotein .beta.-1,2-N-acetylglucosaminyltransferase, .alpha.-1,6-mannosyl-glycoprotein .beta.-1,2-N-acetylglucosaminyltransferase, .beta.-1,4-mannosyl-glycoprotein .beta.-1,2-xylosyltransferase and glycoprotein 3-.alpha.-L-fucosyltransferase in the Golgi fraction of suspension-cultured ***cells*** of sycamore (A. pseudoplatanus L.) were measured using fluorescence-labeled oligosaccharides as acceptor substrates for these transferase reactions. The structures of the pyridylaminated oligosaccharides produced by these reactions analyzed by two-dimensional sugar mapping using high-performance liq. chromatog. A biosynthetic pathway for xylose contg. N-linked oligosaccharides in ***plant*** glycoproteins was discussed.

L7 ANSWER 36 OF 36 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2003:462501 BIOSIS <<LOGINID::20070409>>
DN PREV200300462501
TI Two closely related forms of UDP-GlcNAc: alpha6-D-mannoside beta1,2-N-***acetylglucosaminyltransferase*** ***II*** occur in the clawed frog Xenopus laevis.
AU Mucha, Jan; Svoboda, Barbara; Kappel, Sonja; Strasser, Richard; Bencur, Peter; Froehwein, Ulrike; Schachter, Harry; Mach, Lukas [Reprint Author]; Gloessl, Josef
CS Zentrum fuer Angewandte Genetik, Universitaet fuer Bodenkultur Wien, Muthgasse 18, A-1190, Wien, Austria
lukas.mach@boku.ac.at
SO Glycoconjugate Journal, (March 2002 (2003)) Vol. 19, No. 3, pp. 187-195. print.
ISSN: 0282-0080 (ISSN print).
DT Article
LA English
OS DDBJ-AJ517298; EMBL-AJ517298; GenBank-AJ517298; DDBJ-X89002; EMBL-X89002;
GenBank-X89002
ED Entered STN: 8 Oct 2003
Last Updated on STN: 8 Oct 2003
AB UDP-GlcNAc:alpha6-D-mannoside beta1,2-N-***acetylglucosaminyltransferase***
*** *** ***II*** (GnT ***II*** ; EC 2.4.1.143) is a medial-Golgi resident enzyme that catalyses an essential step in the biosynthetic pathway leading from high mannose to complex N-linked oligosaccharides. Screening a cDNA library from Xenopus laevis ovary with a human GnT II DNA probe resulted in the isolation of two cDNA clones encoding two closely related GnT II isoenzymes, GnT II-A and GnT II-B. Analysis of the corresponding genomic DNAs revealed that the open reading frame of both X. laevis GnT II genes resides within a single exon. The GnT II-A gene was found to be transcriptionally active in all X. laevis tissues tested. In contrast, expression of the GnT II-B gene was detected only in a limited number of tissues. Both GnT II-A and GnT II-B exhibit a type II transmembrane protein topology with a putative N-terminal cytoplasmic tail of 9 amino acids followed by a transmembrane domain of 18 residues, and a C-terminal luminal domain of 405 residues. The two proteins differ at 28 amino acid positions within their luminal regions. Heterologous expression of soluble forms of the enzymes in ***insect*** ***cells*** showed that GnT II-A and GnT II-B are both catalytically active and exhibit similar specific activities. Both recombinant proteins are modified with N-linked oligosaccharides. N-terminal deletion studies demonstrated that the first 49 amino acid residues are not essential for proper folding and enzymatic activity of X. laevis GnT II.

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FULL ESTIMATED COST 0.90 658.87

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with preparation role
NEWS 4 DEC 18 CA/CAplus patent kind codes updated
NEWS 5 DEC 18 MARPAT to CA/CAplus accession number crossover limit increased
to 50,000
NEWS 6 DEC 18 MEDLINE updated in preparation for 2007 reload
NEWS 7 DEC 27 CA/CAplus enhanced with more pre-1907 records
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NEWS 9 JAN 16 CA/CAplus Company Name Thesaurus enhanced and reloaded
NEWS 10 JAN 16 IPC version 2007.01 thesaurus available on STN
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NEWS 12 JAN 22 CA/CAplus updated with revised CAS roles
NEWS 13 JAN 22 CA/CAplus enhanced with patent applications from India
NEWS 14 JAN 29 PHAR reloaded with new search and display fields
NEWS 15 JAN 29 CAS Registry Number crossover limit increased to 300,000 in multiple databases
NEWS 16 FEB 15 PATDPASPC enhanced with Drug Approval numbers
NEWS 17 FEB 15 RUSSIAPAT enhanced with pre-1994 records
NEWS 18 FEB 23 KOREAPAT enhanced with IPC 8 features and functionality
NEWS 19 FEB 26 MEDLINE reloaded with enhancements
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NEWS 21 FEB 26 TOXCENTER enhanced with reloaded MEDLINE
NEWS 22 FEB 26 IFICDB/IFIPAT/IFIUDB reloaded with enhancements
NEWS 23 FEB 26 CAS Registry Number crossover limit increased from 10,000 to 300,000 in multiple databases

NEWS 24 MAR 15 WPIDS/WPIX enhanced with new FRAGHITSTR display format
NEWS 25 MAR 16 CASREACT coverage extended
NEWS 26 MAR 20 MARPAT now updated daily
NEWS 27 MAR 22 LWPI reloaded
NEWS 28 MAR 30 RDISCLOSURE reloaded with enhancements
NEWS 29 MAR 30 INPADOCDB will replace INPADOC on STN
NEWS 30 APR 02 JICST-EPLUS removed from database clusters and STN

NEWS EXPRESS NOVEMBER 10 CURRENT WINDOWS VERSION IS V8.01c, CURRENT
MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
AND CURRENT DISCOVER FILE IS DATED 25 SEPTEMBER 2006.

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FULL ESTIMATED COST 0.21 0.21

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L1 510 N ACETYLGLUCOSAMINYLTRANSFERASE III OR GNTIII OR GNT III

=> s l1 and (yeast or fungi or insect or plant)
L2 22 L1 AND (YEAST OR FUNGI OR INSECT OR PLANT)

=> dup rem l2
PROCESSING COMPLETED FOR L2
L3 17 DUP REM L2 (5 DUPLICATES REMOVED)

=> d bib abs 1-
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L3 ANSWER 1 OF 17 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2007:114069 CAPLUS <<LOGINID::20070410>>
DN 146:212635
TI Remodeling and glycoconjugation of erythropoietin and other therapeutic polypeptides
IN Defrees, Shawn; Zopf, David A.; Bayer, Robert J.; Hakes, David James; Bowe, Caryn; Chen, Xi
PA USA
SO U.S. Pat. Appl. Publ., 753pp., Cont.-in-part of U.S. Ser. No. 410,945.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 17

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2007027068	A1	20070201	US 2005-530972	20051205
WO 2003031464	A2	20030417	WO 2002-US32263	20021009
WO 2003031464	A3	20060302		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 2004137557	A1	20040715	US 2002-287994	20021105
US 7138371	B2	20061121		
US 2007042458	A1	20070222	US 2003-410945	20030409
WO 2004033651	A2	20040422	WO 2003-US31974	20031008
WO 2004033651	A3	20060330		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRAI US 2001-328523P	P	20011010		
US 2001-344692P	P	20011019		
US 2001-334233P	P	20011128		
US 2001-334301P	P	20011128		
US 2002-387292P	P	20020607		
US 2002-391777P	P	20020625		
US 2002-396594P	P	20020717		
US 2002-404249P	P	20020816		
US 2002-407527P	P	20020828		
WO 2002-US32263	A1	20021009		
US 2002-287994	A2	20021105		
US 2003-360770	B2	20030106		
US 2003-360779	B2	20030219		
US 2003-410945	A2	20030409		
WO 2003-US31974	W	20031008		

AB The invention includes methods and compns. for remodeling a peptide mol., including the addn. or deletion of one or more glycosyl groups to a peptide, and/or the addn. of a modifying group to a peptide. A key feature of the invention is to take a peptide produced by any cell type and generate a core glycan structure on the peptide, following which the glycan structure is then remodeled in vitro to generate a peptide having a glycosylation pattern suitable for therapeutic use in a mammal. The invention includes remodeling and PEGylation of erythropoietin, for use in treating anemia or kidney dialysis patients.

L3 ANSWER 2 OF 17 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN DUPLICATE 1
AN 2007114309 EMBASE <<LOGINID::20070410>>
TI Efficient introduction of a bisecting GlcNAc residue in tobacco N-glycans by expression of the gene encoding human ***N*** - ***acetylglucosaminyltransferase*** ***III*** .
AU Rouwendal G.J.A.; Wuhrer M.; Florack D.E.A.; Koeleman C.A.M.; Deelder A.M.; Bakker H.; Stoopen G.M.; van Die I.; Helsper J.P.F.G.; Hokke C.H.; Bosch D.
CS G.J.A. Rouwendal, Business Unit Bioscience, Plant Research International B.V., Wageningen University and Research Centre, Droevendaalsesteeg 1, 6708 PB Wageningen, Netherlands. gerard.rouwendal@wur.nl
SO Glycobiology, (2007) Vol. 17, No. 3, pp. 334-344. .
Refs: 49
ISSN: 0959-6658 E-ISSN: 1460-2423 CODEN: GLYCE3
CY United Kingdom
DT Journal; Article
FS 029 Clinical Biochemistry
037 Drug Literature Index
LA English
SL English
ED Entered STN: 27 Mar 2007
Last Updated on STN: 27 Mar 2007
AB In this study, we show that introduction of human N-acetylglucosaminyltransferase (***GnT***)- ***III*** gene into tobacco plants leads to highly efficient synthesis of bisected N-glycans. Enzymatically released N-glycans from leaf glycoproteins of wild-type and transgenic ***GnT*** - ***III*** plants were profiled by matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF-MS) in native form. After labeling with 2-aminobenzamide, profiling was performed using normal-phase high-performance liquid chromatography with fluorescence detection, and glycans were structurally characterized by MALDI-TOF/TOF-MS and reverse-phase nano-liquid chromatography-MS/MS. These analyses revealed that most of the complex-type N-glycans in the plants expressing ***GnT*** - ***III*** were bisected and carried at least two terminal N-acetylglucosamine (GlcNAc) residues in contrast to wild-type plants, where a considerable proportion of N-glycans did not contain GlcNAc residues at the nonreducing end. Moreover, we have shown that the majority of N-glycans of an antibody produced in a ***plant*** expressing ***GnT*** - ***III*** is also bisected. This might improve the efficacy of therapeutic antibodies produced in this type of transgenic ***plant*** . .COPYRGT. 2007 Oxford University Press.

L3 ANSWER 3 OF 17 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN DUPLICATE 2
AN 2006242628 EMBASE <<LOGINID::20070410>>
TI Influence of variable N-glycosylation on the cytolytic potential of chimeric CD19 antibodies.
AU Barbin K.; Stieglmaier J.; Saul D.; Stieglmaier K.; Stockmeyer B.; Pfeiffer M.; Lang P.; Fey G.H.
CS Dr. G.H. Fey, Department of Genetics, University of Erlangen-Nuremberg, Staudtstrasse 5, D 91058 Erlangen, Germany. gfey@biologie.uni-erlangen.de
SO Journal of Immunotherapy, (2006) Vol. 29, No. 2, pp. 122-133. .
Refs: 50
ISSN: 1524-9557 CODEN: JOIME7

PUI 0000237120060300000002
CY United States
DT Journal; Article
FS 016 Cancer
025 Hematology
026 Immunology, Serology and Transplantation
037 Drug Literature Index
LA English
SL English
ED Entered STN: 22 Jun 2006
Last Updated on STN: 22 Jun 2006
AB To investigate the influence of N-linked oligosaccharides at asparagines-297 on the cytolytic potential of chimeric CD19 antibodies, three distinct variants were generated by production in different expression systems. The same chimeric CD19 antibody was produced in Sf21 ***insect*** cells, human 293 T cells, and 293 T cells expressing a co-transfected .beta.1,4- ***N*** - ***acetylglucosaminyltransferase***** *** * ***III*** (***GnTIII***). The N-glycan structures and the cytolytic potential of the antibodies produced in these three systems were directly compared. After expression in ***insect*** cells, the antibody carried paucimannosidic N-linked oligosaccharides, distinct from the complex biantennary carbohydrate moieties attached to the product from human cells. After co-expression with ***GnTIII*** in human cells, the antibody carried an eightfold greater percentage of oligosaccharides with a bisecting N-acetylglucosamine (78.7% versus 9.6%) and a 30-fold increased proportion of bisecting, defucosylated oligosaccharides (15.9% versus 0.5%). The ***insect*** cell product triggered stronger antibody-dependent cellular cytotoxicity (ADCC) of a human leukemia-derived cell line than the product from non-re-engineered 293 T cells and was equally effective at 50- to 100-fold lower concentrations. The antibody from glyco-engineered 293 T cells had comparable lytic activity as the ***insect*** cell product. Both mediated significant ADCC at lower effector-to-target cell ratios than the antibody from non-re-engineered 293 T cells, and both were highly effective against primary blasts from pediatric leukemia patients. The data demonstrate the influence of the N-glycosylation pattern on the ADCC activity of chimeric CD19 antibodies and point to the importance of suitable expression systems for the production of highly active therapeutic antibodies. Copyright .COPYRGT. 2006 by Lippincott Williams & Wilkins.

L3 ANSWER 4 OF 17 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2005:1154698 CAPLUS <<LOGINID::20070410>>
DN 143:433718
TI Genetically engineered ***yeast*** for production of human-like glycoproteins with terminal galactose residues
IN Davidson, Robert; Gerngross, Tillman; Wildt, Stefan; Choi, Byung-Kwon; Nett, Juergen; Bobrowicz, Piotr; Hamilton, Stephen
PA Glycofi, Inc., USA
SO PCT Int. Appl., 120 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 25

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2005100584	A2	20051027	WO 2005-IB51249	20050415
WO 2005100584	A3	20061221		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2005233387	A1	20051027	AU 2005-233387	20050415
CA 2562772	A1	20051027	CA 2005-2562772	20050415
EP 1737969	A2	20070103	EP 2005-732293	20050415
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, LV, MK, YU				
PRAI US 2004-562424P	P	20040415		
WO 2005-IB51249	W	20050415		

AB The invention provides a lower eukaryotic host cell producing human-like glycoproteins characterized as having a terminal .beta.-galactose residue and essentially lacking fucose and sialic acid residues. The invention also provides methods and compns., including genetic vectors, for catalyzing the transfer of a galactose residue from UDP-galactose onto an acceptor substrate in a recombinant lower eukaryotic host cell. In addn. to a UDP-Gal:.beta.GlcNAc .beta.-1,4-galactosyltransferase, expression of UDP-galactose transporter(s), a UDP-specific diphosphatase, and UDP-galactose-4-epimerase, galactokinase, or galactose-1-phosphate uridyltransferase activities allow transfer of galactose residues onto preferred acceptor substrates for use as therapeutic glycoproteins. The invention claims polypeptide sequences for gene galE UDP-galactose C4 epimerase enzyme and conserved motifs. Methods of the invention can be applied to therapeutic glycoproteins such as erythropoietin, cytokines, blood coagulation factors, Igs, growth factors, or plasminogen. The examples provide maps of integrating plasmid vectors encoding human GalTI, S. pombe gene galE epimerase, and D. melanogaster gene UGT UDP-galactose transporter. The secreted kringle 3 (K3) domain of plasminogen was the reporter protein for glycosylation in transformed Pichia pastoris strains. N-linked glycans obtained from K3 were analyzed by MALDI-TOF mass spectrometry. A P. pastoris strain with och1 and alg3 gene deletions, active fusion constructs of mouse mannosidase IB and human GnTI, the Kluyveromyces lactis UDP-GlcNAc transporter gene, and a human GalTI gene leader fusion construct had approx. 10-20% of GlcNAc2Man3GlcNAc2 N-glycans on K3 converted to GalGlcNAc2Man3GlcNAc2 and 1-2% to Gal2GlcNAc2Man3GlcNAc2. When a strain with the same genotype was also transformed with the Saccharomyces cerevisiae epimerase gene GAL10 under control of the PMAI promoter, about 2/3 of the N-glycans released from K3 contained an addnl. hexose residue (HexGlcNAcMan5GlcNAc2) that could be removed by sol. .beta.-1,4-galactosidase.

L3 ANSWER 5 OF 17 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2005:409684 CAPLUS <<LOGINID::20070410>>
DN 142:458111
TI Production of human glycosylated proteins in transgenic insects
IN Jarvis, Donald; Van Beek, Nikolai; Fraser, Malcolm
PA Chesapeake Perl, Inc., USA
SO PCT Int. Appl., 81 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2005042753	A1	20050512	WO 2004-US35553	20041028
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2007067855	A1	20070322	US 2006-577528	20060428
PRAI US 2003-514741P	P	20031028		
WO 2004-US35553	W	20041028		

AB The invention provides transgenic insects, or progeny thereof, whose cells contain at least one integrated nucleic acid encoding two or more N-glycosylation enzymes that are used to glycosylate a heterologous protein with a mammalianized (humanized) pattern. Specifically, the invention provides transgenic insects transformed with vectors encoding: (a) various N-acetylglucosaminyltransferases (GlcNAc-Ts), sialyltransferases (.alpha.2,6-sialyltransferase and .alpha.2,3-sialyltransferase), sialic acid synthase and CMP-sialic acid synthetase; (b) various auxiliary glycosylation proteins (such as transport proteins); and (c) a heterologous protein of interest (such as antibody, receptor, vaccine). The invention relates that said glycosylation enzymes are expressed and used to produce glycosylated proteins of interest. The invention also provides methods for producing said humanized glycosylated proteins using transgenic ***insect*** larva and baculovirus-based or transposon-based vectors carrying said nucleic acids. The invention further provides a library of different types of TRANSPILLAR larva expressing different glycoproteins of interest. The invention briefly discussed the use of said transgenic ***insect*** cells in manufg. authentic human-type glycoproteins for therapeutic applications (no data).
RE.CNT 10 THERE ARE 10 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 6 OF 17 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2005:1028015 CAPLUS <<LOGINID::20070410>>
DN 143:300313
TI N-acetylglucosamintransferase III expression in genetically modified lower eukaryotes
IN Bobrowicz, Piotr; Hamilton, Stephen R.; Gerngross, Tilman U.; Wildt, Stefan; Choi, Byung-Kwon; Nett, Juergen Hermann; Davidson, Robert C.
PA USA
SO U.S. Pat. Appl. Publ., 163 pp., Cont.-in-part of U.S. Ser. No. 371,877.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 25

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2005208617	A1	20050922	US 2003-680963	20031007
US 2002137134	A1	20020926	US 2001-892591	20010627
US 7029872	B2	20060418		
EP 1522590	A1	20050413	EP 2004-25648	20010627
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY, TR				
WO 2003056914	A1	20030717	WO 2002-US41510	20021224
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,				

LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
US 2004018590 A1 20040129 US 2003-371877 20030220
AU 2004213859 A1 20040902 AU 2004-213859 20040220
AU 2004213868 A1 20040902 AU 2004-213868 20040220
CA 2516520 A1 20040902 CA 2004-2516520 20040220
CA 2516550 A1 20040902 CA 2004-2516550 20040220
WO 2004074458 A2 20040902 WO 2004-US5128 20040220
WO 2004074458 A3 20041229
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU; CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
WO 2004074461 A2 20040902 WO 2004-US5191 20040220
WO 2004074461 A3 20050317
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH. CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
EP 1597381 A2 20051123 EP 2004-713388 20040220
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK
EP 1599595 A2 20051130 EP 2004-713412 20040220
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK
JP 2006518597 T 20060817 JP 2006-503757 20040220
JP 2006518600 T 20060817 JP 2006-503776 20040220
US 2007037248 A1 20070215 US 2006-546101 20060803
PRAI US 2000-214358P P 20000628
US 2000-215638P P 20000630
US 2001-279997P P 20010330
US 2001-892591 A2 20010627
US 2001-344169P P 20011227
WO 2002-US41510 A2 20021224
US 2003-371877 A2 20030220
EP 2001-954606 A3 20010627
WO 2002-US241510 W 20021224
US 2003-680963 A 20031007
WO 2004-US5128 A 20040220
WO 2004-US5191 A 20040220
US 2005-500240 A2 20050323

AB The present invention relates to eukaryotic host cells having modified oligosaccharides which may be modified further by heterologous expression of a set of glycosyltransferases, sugar transporters and mannosidases to become host-strains for the prodn. of mammalian, e.g., human therapeutic glycoproteins. The present invention relates to methods and compns. by which non-human eucaryotic cells, such as ***fungi*** or other eukaryotic cells, can be genetically modified to produce glycosylated proteins (glycoproteins) having patterns of glycosylation similar to those of glycoproteins produced by animal cells, esp. human cells, which are useful as human or animal therapeutic agents. The process provides an engineered host cell which can be used to express and target any desirable gene(s) involved in glycosylation. Host cells with modified lipid-linked oligosaccharides are created or selected. N-glycans made in the engineered host cells exhibit ***GnTIII*** activity, which produce bisected N-glycan structures and may be modified further by heterologous expression of one or more enzymes, e.g., glycosyltransferases, sugar transporters and mannosidases, to yield human-like glycoproteins. For the prodn. of therapeutic proteins, this method may be adapted to engineer cell lines in which any desired glycosylation structure may be obtained.

L3 ANSWER 7 OF 17 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2005:122585 CAPLUS <<LOGINID::20070410>>
DN 142:217398
TI Cell-free in vitro glycoconjugation of interleukin 2 as therapeutic agent against cancer and AIDS in mammal and human
IN Defrees, Shawn; Zopf, David; Bayer, Robert; Bowe, Caryn; Hakes, David; Chen, Xi
PA Neose Technologies, Inc., USA
SO U.S. Pat. Appl. Publ., 750 pp., Cont.-in-part of U.S. Ser. No. 360,779.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 17

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2005031584	A1	20050210	US 2003-410980	20030409
WO 2003031464	A2	20030417	WO 2002-US32263	20021009
WO 2003031464	A3	20060302		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
US 2004137557 A1 20040715 US 2002-287994 20021105
US 7138371 B2 20061121
AU 2004236174 A1 20041118 AU 2004-236174 20040409
CA 2522345 A1 20041118 CA 2004-2522345 20040409
WO 2004099231 A2 20041118 WO 2004-US11494 20040409
WO 2004099231 A3 20060316
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
EP 1615945 A2 20060118 EP 2004-750118 20040409
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, HR
CN 1863458 A 20061115 CN 2004-80015918 20040409
US 2007026485 A1 20070201 US 2006-552896 20060608
PRAI US 2001-328523P P 20011010
US 2001-344692P P 20011019
US 2001-334233P P 20011128
US 2001-334301P P 20011128
US 2002-387292P P 20020607
US 2002-391777P P 20020625
US 2002-396594P P 20020717
US 2002-404249P P 20020816
US 2002-407527P P 20020828
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US 2002-287994 A2 20021105
US 2003-360770 A2 20030106
US 2003-360779 A2 20030219
US 2003-410897 A 20030409
US 2003-410913 A 20030409
US 2003-410930 A 20030409
US 2003-410945 A 20030409
US 2003-410962 A 20030409
US 2003-410980 A 20030409
US 2003-410997 A 20030409
US 2003-411012 A 20030409
US 2003-411026 A 20030409
US 2003-411037 A 20030409
US 2003-411043 A 20030409
US 2003-411044 A 20030409
US 2003-411049 A 20030409
WO 2004-US11494 A 20040409

AB The invention includes methods and compns. for remodeling a peptide mol., including the addn. or deletion of one or more glycosyl groups to a peptide, and/or the addn. of a modifying group to a peptide. The method uses enzyme to remove or add phosphate, sulfate, carboxylate and/or ester group-contg. saccharide to interleukin 2 peptide, and then conjugate the saccharide-linked interleukin 2 with modifying group such as polymer, therapeutic moiety, detectable label, toxin, radioisotope, targeting moiety and peptide. The saccharide group comprises monosaccharyl, oligosaccharyl, glycosyl, truncated glycan, mannosyl, GlcNAc, xylosyl, sialyl, galactosyl, glucosyl or GalNAc. The enzyme for the saccharide addn. or removal is a prokaryotic or eukaryotic glycosyltransferase selected from sialyltransferase, galactosyltransferase, glucosyltransferase, GalNAc transferase, GlcNAc transferase, fucosyltransferase, mannosyltransferase, endo-N-acetylgalactosaminidase, glycosidase, sialidase, mannosidase, etc. The substrate is a nucleotide sugar such as UDP-glucose, UDP-galactose, UDP-galactosamine, UDP-glucosamine, UDP-N-acetylgalactosamine, UDP-N-acetylglucosamine, GDP-mannose, GDP-fucose, CMP-sialic acid and CMP-NeuAc.

L3 ANSWER 8 OF 17 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
DUPLICATE 3
AN 2006:125729 BIOSIS <<LOGINID::20070410>>
DN PREV200600113050
TI Control of recombinant monoclonal antibody effector functions by Fc N-glycan remodeling in vitro.
AU Hodoniczky, Jason; Zheng, Yuan Zhi; James, David C. [Reprint Author]
CS Univ Queensland, Sch Engn, St Lucia, Qld 4072, Australia
davidj@cheque.uq.edu.au
SO Biotechnology Progress, (NOV-DEC 2005) Vol. 21, No. 6, pp. 1644-1652.
CODEN: BIPRET. ISSN: 8756-7938.
DT Article
LA English
ED Entered STN: 15 Feb 2006
Last Updated on STN: 15 Feb 2006

AB N-Glycans at Asn(297) in the Fc domain of IgG molecules are required for Fc receptor-mediated effector functions such as antibody-dependent cell-mediated cytotoxicity (ADCC) and complement-dependent cytotoxicity (CDC). In this study we have specifically remodeled the Fc N-glycans of intact recombinant IgG(1) therapeutic monoclonal antibody (Mab) products, Rituxan and Herceptin, with a soluble recombinant rat beta-1,4- ***N*** - ***acetylglucosaminyltransferase*** ***III*** (rGnTIII) produced by baculovirus-infected ***insect*** cells. N-Glycan remodeling in vitro permitted a controlled and selective transfer of a bisecting beta 1,4-linked GlcNAc to the core beta-linked mannose of degalactosylated Mab N-glycans to yield Mabs varying in bisecting GlcNAc: content from 31% to 85%. This was confirmed by analysis of N-glycans by both normal phase HPLC and MALDI-MS, the latter yielding the expected mass increase of 203.2 Da with no other oligosaccharide modifications evident. ADCC of remodeled Rituxan and Herceptin Mabs was determined using peripheral blood mononuclear cells as effectors and either CD20(+) (SKW6.4 and SU-DHL-4) or Her2(+) (SKBR-3) target cells, respectively. A conserved 10-fold increase in ADCC was observed for both remodeled therapeutic Mabs with high (> 80%) bisecting GlcNAc content. In contrast, although the presence of a bisecting GlcNAc had minimal effect on CDC, degalactosylation of Rituxan reduced CDC by approximately half, relative to unmodified (variably galactosylated) control Mab. In summary, our data suggests that in vitro remodeling of therapeutic Mab Fc N-glycans may be utilized to control the therapeutic efficacy of Mabs in vivo and to offer a more "humanized" glycoform profile for recombinant Mab products.

L3 ANSWER 9 OF 17 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:720587 CAPLUS <<LOGINID::20070410>>
DN 141:237748
TI ***N*** - ***acetylglucosaminyltransferase*** ***III*** and other N-glycan-processing enzymes expressed in lower eukaryotes for the biosynthesis of human-like oligosaccharide structures in glycoproteins
IN Bobrowicz, Piotr; Hamilton, Stephen R.; Gerngross, Tillman U.; Wildt, Stefan; Choi, Byung-Kwon; Nett, Juergen Hermann; Davidson, Robert C.
PA USA
SO PCT Int. Appl., 193 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 25

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2004074458	A2	20040902	WO 2004-US5128	20040220
WO 2004074458	A3	20041229		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2004018590	A1	20040129	US 2003-371877	20030220
US 2005208617	A1	20050922	US 2003-680963	20031007
AU 2004213859	A1	20040902	AU 2004-213859	20040220
CA 2516520	A1	20040902	CA 2004-2516520	20040220
EP 1599595	A2	20051130	EP 2004-713412	20040220
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JP 2006518597	T	20060817	JP 2006-503757	20040220
PRAI US 2003-371877	A	20030220		
US 2003-680963	A	20031007		
US 2000-214358P	P	20000628		
US 2000-215638P	P	20000630		
US 2001-279997P	P	20010330		
US 2001-892591	A2	20010627		
US 2001-344169P	P	20011227		
WO 2002-US41510	A2	20021224		
WO 2004-US5128	A	20040220		

AB The present invention relates to eukaryotic host cells having modified oligosaccharides which may be modified further by heterologous expression of a set of glycosyltransferases, sugar transporters, and mannosidases to become host-strains for the prodn. of mammalian, e.g., human therapeutic glycoproteins. The process provides an engineered host cell such as Pichia pastoris which can be used to express and target any desirable gene(s) involved in glycosylation. Host cells with modified lipid-linked oligosaccharides are created or selected. N-glycans made in the engineered host cells exhibit ***N*** - ***acetylglucosaminyltransfera*** *** se*** ***III*** (***GnTIII***) activity, which produce bisected N-glycan structures and may be modified further by heterologous expression of one or more enzymes, e.g., glycosyltransferases, sugar transporters and mannosidases, to yield human-like glycoproteins. For the prodn. of therapeutic proteins, this method may be adapted to engineer cell lines in which any desired glycosylation structure may be obtained.

L3 ANSWER 10 OF 17 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:634026 CAPLUS <<LOGINID::20070410>>
DN 141:172878
TI Engineering of glycosylation profile of antibody Fc region to increase Fc receptor binding affinity and effector function for treating cancer
IN Umana, Pablo; Bruenker, Peter; Ferrara, Claudia; Suter, Tobias
PA Glycart Biotechnology Ag, Switz.
SO PCT Int. Appl., 231 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2004065540	A2	20040805	WO 2004-IB844	20040122
WO 2004065540	A3	20050324		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI				
AU 2004205802	A1	20040805	AU 2004-205802	20040122
CA 2513797	A1	20040805	CA 2004-2513797	20040122
US 2004241817	A1	20041202	US 2004-761435	20040122
EP 1587921	A2	20051026	EP 2004-704310	20040122
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
CN 1761746	A	20060419	CN 2004-80007564	20040122
JP 2006516893	T	20060713	JP 2006-500338	20040122
IN 2005KN01628	A	20060901	IN 2005-KN1628	20050816
NO 2005003872	A	20051021	NO 2005-3872	20050818
PRAI US 2003-441307P	P	20030122		
US 2003-491254P	P	20030731		
US 2003-495142P	P	20030815		
WO 2004-IB844	W	20040122		

AB The present invention relates to nucleic acid mols., including fusion constructs, having catalytic activity and the use of same in glycosylation engineering of host cells to generate polypeptides with improved therapeutic properties, including antibodies with increased Fc receptor binding and increased effector function. The engineered proteins or antibodies comprise Golgi localization domain of Golgi resident polypeptide such as .beta.(1,4)- ***N*** - ***acetylglucosaminyltransferase*** ***III*** , .beta.(1,4)-galactosyltransferase, mannosidase II, .beta.(1,2)-N-acetylglucosaminyltransferase I, .beta.(1,2)-N-acetylglucosaminyltransferase II, mannosidase I, .alpha.-mannosidase II, and .alpha.1-6 core fucosyltransferase. The effector function includes Fc-mediated cellular cytotoxicity of NK cells, macrophage, polymorphonuclear cells and monocytes; signaling of apoptosis induction; maturation of dendritic cells; or T cell priming. The engineered antibodies include antibodies or humanized antibodies specific to human neuroblastoma, renal cell carcinoma, colon carcinoma, breast carcinoma, lung carcinoma, 17-1A antigen, CD20, CD22, CD30, CD40, PSMA, EGFR, PSCA, HLA-DR, MUC1, EpCAM, etc.

L3 ANSWER 11 OF 17 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:80241 CAPLUS <<LOGINID::20070410>>
DN 140:158561
TI Combinatorial DNA library of mammalian glycosylation enzyme genes used for producing modified n-glycans in lower eukaryotes
IN Gerngross, Tillman U.; Wildt, Stefan; Choi, Byung-Kwon; Nett, Juergen Hermann; Bobrowicz, Piotr; Hamilton, Stephen R.; Davidson, Robert C.
PA USA
SO U.S. Pat. Appl. Publ., 97 pp., Cont.-in-part of U.S. Ser. No. 892,591.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 25

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2004018590	A1	20040129	US 2003-371877	20030220
US 2002137134	A1	20020926	US 2001-892591	20010627
US 7029872	B2	20060418		
EP 1522590	A1	20050413	EP 2004-25648	20010627
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US 2004230042	A1	20041118	US 2003-616082	20030708
US 2005208617	A1	20050922	US 2003-680963	20031007
US 2004171826	A1	20040902	US 2003-695243	20031027
AU 2004213859	A1	20040902	AU 2004-213859	20040220
AU 2004213860	A1	20040902	AU 2004-213860	20040220
AU 2004213861	A1	20040902	AU 2004-213861	20040220
AU 2004213868	A1	20040902	AU 2004-213868	20040220
AU 2004213869	A1	20040902	AU 2004-213869	20040220
CA 2516440	A1	20040902	CA 2004-2516440	20040220
CA 2516520	A1	20040902	CA 2004-2516520	20040220
CA 2516527	A1	20040902	CA 2004-2516527	20040220
CA 2516544	A1	20040902	CA 2004-2516544	20040220
CA 2516550	A1	20040902	CA 2004-2516550	20040220
WO 2004074458	A2	20040902	WO 2004-US5128	20040220
WO 2004074458	A3	20041229		
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WO 2004074497	A2	20040902	WO 2004-US5131	20040220
WO 2004074497	A3	20041202		

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WO 2004074498 A2 20040902 WO 2004-US5132 20040220
WO 2004074498 A3 20050623
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WO 2004074461 A2 20040902 WO 2004-US5191 20040220
WO 2004074461 A3 20050317
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WO 2004074499 A2 20040902 WO 2004-US5244 20040220
WO 2004074499 A3 20050127
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EP 1597379 A2 20051123 EP 2004-713369 20040220
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EP 1597380 A2 20051123 EP 2004-713372 20040220
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EP 1597381 A2 20051123 EP 2004-713388 20040220
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EP 1599595 A2 20051130 EP 2004-713412 20040220
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK
EP 1599596 A2 20051130 EP 2004-713437 20040220
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK
JP 2006518597 T 20060817 JP 2006-503757 20040220
JP 2006518598 T 20060817 JP 2006-503759 20040220
JP 2006518599 T 20060817 JP 2006-503760 20040220
JP 2006518600 T 20060817 JP 2006-503776 20040220
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US 2006040353 A1 20060223 US 2005-108088 20050415
US 2006024304 A1 20060202 US 2005-187196 20050721
US 2006029604 A1 20060209 US 2005-187229 20050721
US 2006034828 A1 20060216 US 2005-187066 20050721
US 2006034830 A1 20060216 US 2005-187113 20050721
IN 2005KN01844 A 20061103 IN 2005-KN1844 20050916
US 2006078963 A1 20060413 US 2005-240432 20050930
US 2006177898 A1 20060810 US 2005-249061 20051011
US 2006148035 A1 20060706 US 2005-271235 20051110
US 2006257399 A1 20061116 US 2005-317191 20051222
US 2006286637 A1 20061221 US 2006-429672 20060505
US 2007037248 A1 20070215 US 2006-546101 20060803
PRAI US 2000-214358P P 20000628
US 2000-215638P P 20000630
US 2001-279997P P 20010330
US 2001-892591 A2 20010627
EP 2001-954606 A3 20010627
US 2001-344169P P 20011227
WO 2002-US241510 W 20021224
WO 2002-US41510 A2 20021224
US 2003-371877 A2 20030220
US 2003-616082 A 20030708
US 2003-680963 A 20031007
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WO 2004-US5132 W 20040220
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US 2004-554139P P 20040317
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US 2004-639542P P 20041223
US 2004-639629P P 20041223
US 2004-639630P P 20041223
US 2004-639631P P 20041223
US 2004-639657P P 20041223
US 2004-639698P P 20041223
US 2005-84624 A2 20050317
US 2005-500240 A2 20050323
US 2005-108088 A2 20050415

AB The present invention relates to use of combinatorial DNA library of mammalian glycosylation enzyme genes for producing modified n-glycans in lower eukaryotes. The invention provides nucleic acid mols. and combinatorial libraries which can be used to successfully target and express mammalian enzymic activities such as those involved in glycosylation to intracellular compartments in a eukaryotic host cell. Heterologous expression of a set of glycosyltransferases, sugar transporters and mannosidases in eukaryotic host cells enables oligosaccharide modification and the development of host-strains for the prodn. of mammalian glycoproteins. The process provides an engineered host cell which can be used to express and target any desirable gene(s) involved in glycosylation. Host cells with modified oligosaccharides are created or selected. N-glycans made in the engineered host cells have a Man 5 GlcNAc 2 core structure which may then be modified further by heterologous expression of one or more enzymes, e.g., glycosyltransferases, sugar transporters and mannosidases, to yield human-like glycoproteins. With the primary goal of prodn. of human therapeutic glycoproteins, this method may be adapted to engineer cell lines in which any desired glycosylation structure may be obtained.

L3 ANSWER 12 OF 17 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2003:757842 CAPLUS <<LOGINID::20070410>>
DN 139:272047
TI Mammalian UDP-N-acetylglucosamine:.beta.-D-mannoside .beta.(1,4)-N-acetylglucosaminyltransferase (***GnTIII***) expression in plants
IN Bakker, Hendrikus Antonius Cornelus; Florack, Dionisius Elisabeth Antonius; Bosch, Hendrik Jan
PA Plant Research International B.V., Neth.
SO PCT Int. Appl., 122 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2003078614	A2	20030925	WO 2003-IB1562	20030318
WO 2003078614	A3	20040108		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2478294	A1	20030925	CA 2003-2478294	20030318
AU 2003219402	A1	20030929	AU 2003-219402	20030318
EP 1485492	A2	20041215	EP 2003-715213	20030318
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
CN 1665934	A	20050907	CN 2003-806546	20030318
JP 2005528092	T	20050922	JP 2003-576608	20030318
NZ 534881	A	20060929	NZ 2003-534881	20030318
US 2005223430	A1	20051006	US 2004-508166	20040917
PRAI US 2002-365769P	P	20020319		
US 2002-368047P	P	20020326		
US 2002-365769	P	20020319		
US 2002-368047	P	20020326		
WO 2003-IB1562	W	20030318		

AB The invention relates to the field of glycoprotein processing in transgenic plants used as cost efficient and contamination safe factories for the prodn. of recombinant biopharmaceutical proteins or pharmaceutical comps. comprising these glycoproteins. The invention provides a ***plant*** comprising a functional mammalian enzyme providing mammalian UDP-N-acetylglucosamine:.beta.-D-mannoside .beta.(1,4)-N-acetylglucosaminyltransferase (***GnTIII***) that is normally not present in plants, said ***plant*** addnl. comprising at least a second mammalian protein or functional fragment thereof that is normally not present in plants. The invention further relates to a hybrid protein comprising the catalytic site of ***GnTIII*** and transmembrane domain of Golgi app. and/or endoplasmic reticulum (ER) protein or modified ***GnTIII*** comprising ER retention signals and its use in producing glycoproteins with oligosaccharides that lack immunogenic xylose and fucose residues. Thus, the human gene for ***GnTIII*** was cloned, and a C-terminal c-myc tag for anal. of expression of the tagged fusion protein was provided and the whole was placed under the control of ***plant*** regulatory elements for introduction in maize. ***GnTIII*** is expressed in plants and expression results in bisected oligosaccharide structures on endogenous ***plant*** glycoproteins.

The amt. of N-glycans contg. at lease two GlcNAc residues more than doubled compared to those found in normal maize plants. Expression of ***GnTIII*** also resulted in a significant redn. of complex type N-glycan degrdn. products as apparent from matrix-assisted laser desorption ionization time-of-flight (MALDI-TOF) analyses of the isolated glycans of endogenous ***plant*** glycoproteins. These data suggest that expression of ***GnTIII*** in maize resulting in the introduction of bisected structures on N-glycans protects the glycans from degrdn. by .beta.-N-acetylhexosaminidases.

L3 ANSWER 13 OF 17 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2003:551280 CAPLUS <<LOGINID::20070410>>
DN 139:112733
TI Methods for production of recombinant glycoproteins with mammalian-type carbohydrate structures and their use for production of immunoglobulins
IN Wildt, Stefan; Miele, Robert Gordon; Nett, Juergen Hermann; Davidson, Robert C.
PA Glycofi, Inc., USA
SO PCT Int. Appl., 125 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 25

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2003056914	A1	20030717	WO 2002-US41510	20021224
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2471551	A1	20030717	CA 2002-2471551	20021224
AU 2002358296	A1	20030724	AU 2002-358296	20021224
EP 1467615	A1	20041020	EP 2002-792535	20021224
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JP 2005514021	T	20050519	JP 2003-557288	20021224
US 2005170452	A1	20050804	US 2003-500240	20021224
US 2004230042	A1	20041118	US 2003-616082	20030708
US 2005208617	A1	20050922	US 2003-680963	20031007
US 2006040353	A1	20060223	US 2005-108088	20050415
US 2006024292	A1	20060202	US 2005-187065	20050721
US 2006029604	A1	20060209	US 2005-187229	20050721
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US 2006034830	A1	20060216	US 2005-187113	20050721
US 2006286637	A1	20061221	US 2006-429672	20060505
US 2007037248	A1	20070215	US 2006-546101	20060803
PRAI US 2001-344169P	P	20011227		
US 2000-214358P	P	20000628		
US 2000-215638P	P	20000630		
US 2001-279997P	P	20010330		
US 2001-892591	A2	20010627		
WO 2002-US241510	W	20021224		
WO 2002-US41510	W	20021224		
US 2003-371877	A2	20030220		
US 2003-680963	A	20031007		
WO 2004-US5191	W	20040220		
US 2004-554139P	P	20040317		
US 2004-562424P	P	20040415		
US 2004-589913P	P	20040721		
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US 2004-590011P	P	20040721		
US 2004-590030P	P	20040721		
US 2004-590051P	P	20040721		
US 2004-590052P	P	20040721		
US 2004-639657P	P	20041223		
US 2004-639698P	P	20041223		
US 2005-84624	A2	20050317		
US 2005-500240	A2	20050323		
US 2005-108088	A2	20050415		

AB The present invention relates to host cells having modified lipid-linked oligosaccharides which may be modified further by heterologous expression of a set of glycosyltransferases, sugar transporters and mannosidases to become host-strains for the prodn. of mammalian, e.g., human therapeutic glycoproteins. The process provides an engineered host cell which can be used to express and target any desirable gene(s) involved in glycosylation. Host cells with modified lipid-linked oligosaccharides are created or selected. N-glycans made in the engineered host cells have a GlcNAcMan3GlcNAc2 core structure which may then be modified further by heterologous expression of one or more enzymes, e.g., glycosyltransferases, sugar transporters and mannosidases, to yield human-like glycoproteins. For the prodn. of therapeutic proteins, this method may be adapted to engineer cell lines in which any desired glycosylation structure may be obtained. The invention specifically claims use of nucleic acid sequences for gene ALG3 from Pichia pastoris. The ALG3 gene encodes the enzyme which transfers a mannose residue to the Man5-GlcNac2-PP-Dol precursor. The invention also claims use of genetically engineered host cells for recombinant prodn. of Igs. In examples of the invention, a Pichia pastoris strain with deletions of genes alg3 and och1 was constructed. This strain was transformed with the Kringle 3 domain of human plasminogen as a glycosylation substrate. Mass spectrometric anal. of N-glycans isolated from the kringle 3 glycoproteins showed GlcNAcMan3GlcNAc2 and GlcNAcMan4GlcNAc2 structures which could be further modified in vitro. Addn. of N-acetylglucosamine to GlcNAcMan3GlcNAc2 by N-acetylglucosaminyltransferases II and III yields a "bisected" N-glycan, GlcNAc3Man3GlcNAc2, which has been implicated in greater antibody-dependent cellular cytotoxicity. Methods of the invention can be used to engineer a ***yeast*** strain capable of producing glycoproteins with bisected N-glycans and expressing Ig mols. with bisected N-glycans attached to asparagine residue 297 in the CH2 portion.

RE.CNT 15 THERE ARE 15 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 14 OF 17 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:10683 CAPLUS <<LOGINID::20070410>>
DN 136:80826
TI Methods for producing modified glycoproteins in lower eukaryotes expressing mammalian genes for enzymes of glycosylation
IN Gerngross, Tillman U.
PA Glycofi, Inc., USA
SO PCT Int. Appl., 51 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 25

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2002000879	A2	20020103	WO 2001-US20553	20010627
WO 2002000879	A3	20020906		
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CA 2412701	A1	20020103	CA 2001-2412701	20010627
EP 1297172	A2	20030402	EP 2001-954606	20010627
EP 1297172	B1	20051109		
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JP 2004501642	T	20040122	JP 2002-506194	20010627
NZ 523476	A	20040430	NZ 2001-523476	20010627
EP 1522590	A1	20050413	EP 2004-25648	20010627
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AT 309385	T	20051115	AT 2001-954606	20010627
ES 2252261	T3	20060516	ES 2001-1954606	20010627
PRAI US 2000-214358P	P	20000628		
US 2000-215638P	P	20000630		
US 2001-279997P	P	20010330		
EP 2001-954606	A3	20010627		
WO 2001-US20553	W	20010627		

AB Cell lines having genetically modified glycosylation pathways that allow them to carry out a sequence of enzymic reactions, which mimic the processing of glycoproteins in humans, have been developed. Recombinant proteins expressed in these engineered hosts yield glycoproteins more similar, if not substantially identical to their human counterparts. The lower eukaryotes, which ordinarily produce high-mannose contg. N-glycans, including unicellular and multicellular ***fungi*** are modified to produce N-glycans such as Man5GlcNAc2 or other structures along human glycosylation pathways. This is achieved using a combination of engineering and/or selection of strains which: do not express certain enzymes, such as phospho mannosyltransferase, 1,6-mannosyltransferase, 1,3-mannosyltransferase and 1,2-mannosyltransferase, which create the undesirable complex structures characteristic of the fungal glycoproteins. The expressed exogenous enzymes selected either have optimal activity under the conditions present in the ***fungi*** where activity is desired, or which are targeted to an organelle where optimal activity is achieved. The said engineering and/or selection of strains combinations provide a method for genetically engineering eukaryote expressing multiple exogenous enzymes required to produce "human-like" glycoproteins.

L3 ANSWER 15 OF 17 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2001:680116 CAPLUS <<LOGINID::20070410>>
DN 136:335815
TI Screen for proteins that can interact with glycosyltrdansferases with ***yeast*** two-hybrid system
AU Jiang, Neng-Qun; Zhang, Song-Wen; Zhang, Wei-Jie; Gu, Jian-Xin
CS Sch. Life Sci. Technol., Shanghai Jiatong Univ., Shanghai, 200030, Peop. Rep. China
SO Shanghai Jiaotong Daxue Xuebao (2001), 35(7), 1076-1080
CODEN: SCTPDH; ISSN: 0253-9942
PB Shanghai Jiaotong Daxue Chubanshe
DT Journal
LA Chinese

AB A protein which can interacts with ***N*** - ***acetylglucosaminyltransferase*** ***III*** (***GNT*** ***III***) was obtained from human fetal liver cDNA library with ***yeast*** two hybrid system. This protein is promyelocytic leukemia zinc finger protein (PLZF) which is a transcriptional factor and a member of retinoic acid receptor family. We speculate that PLZF is one of the subjects of enzyme ***GNT*** ***III*** . Also, two proteins which can interact with .beta. 1,4-galactosyltransferase II (GT II) were obtained from human fetal liver cDNA library with ***yeast*** two-hybrid system. One of the two proteins is fibronectin, and this interaction may take part in intercellular conglutination. The other is a protein coded by a new gene.

L3 ANSWER 16 OF 17 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2001:37545 BIOSIS <<LOGINID::20070410>>
DN PREV200100037545
TI Kinetic basis for the donor nucleotide-sugar specificity of beta1,4-***N*** - ***acetylglucosaminyltransferase*** ***III*** .
AU Ikeda, Yoshitaka; Koyota, Souichi; Ihara, Hideyuki; Yamaguchi, Yukihiro; Korekane, Hiroaki; Tsuda, Takeo; Sasai, Ken; Taniguchi, Naoyuki [Reprint author]
CS Department of Biochemistry, Osaka University Medical School, 2-2 Yamadaoka, Suita, Osaka, 565-0871, Japan
proftani@biochem.med.osaka-u.ac.jp
SO Journal of Biochemistry (Tokyo), (Oct., 2000) Vol. 128, No. 4, pp. 609-619. print.
CODEN: JOBIAO. ISSN: 0021-924X.
DT Article
LA English
ED Entered STN: 17 Jan 2001
Last Updated on STN: 12 Feb 2002
AB The kinetic basis of the donor substrate specificity of beta1,4- ***N*** - ***acetylglucosaminyltransferase*** ***III*** (***GnT*** - ***III***) was investigated using a purified recombinant enzyme. The enzyme also transfers GalNAc and Glc moieties from their respective UDP-sugars to an acceptor at rates of 0.1-0.2% of that for GlcNAc, but Gal is not transferred at a detectable rate. Kinetic analyses revealed that these inefficient transfers, which are associated with the specificity of the enzyme, are due to the much lower Vmax values, whereas the Km values for UDP-GalNAc and UDP-Glc differ only slightly from that for UDP-GlcNAc. It was also found that various other nucleotide-Glc derivatives bind to the enzyme with comparable affinities to those of UDP-GlcNAc and UDP-Glc, although the derivatives do not serve as glycosyl donors. Thus, ***GnT*** - ***III*** does not appear to distinguish UDP-GlcNAc from other structurally similar nucleotide-sugars by specific binding in the ground state. These findings suggest that the specificity of ***GnT*** - ***III*** toward the nucleotide-sugar is determined during the catalytic process. This type of specificity may be efficient in preventing a possible mistransfer when other nucleotide-sugars are present in excess over the true donor.

L3 ANSWER 17 OF 17 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 1995:527802 BIOSIS <<LOGINID::20070410>>
DN PREV199598542102
TI Synthesis of pentasaccharide analogues of the N-glycan substrates of ***N*** - ***acetylglucosaminyltransferase*** ***III*** , IV and V using tetrasaccharide precursors and recombinant beta-(1 fwdarw 2)-N-acetylglucosaminyltransferase II.
AU Reck, Folkert; Meinjohanns, Ernst; Tan, Jenny; Grey, Arthur A.; Paulsen, Hans; Schachter, Harry [Reprint author]
CS Res. Inst., Hosp. Sick Children, Toronto, ON M5G 1X8, Canada
SO Carbohydrate Research, (1995) Vol. 275, No. 2, pp. 221-229.
CODEN: CRBRAT. ISSN: 0008-6215.
DT Article
LA English
ED Entered STN: 14 Dec 1995
Last Updated on STN: 27 Jan 1996
AB Recombinant human UDP-GlcNAc:alpha-Man-(1 fwdarw 6)R beta-(1 fwdarw 2)-N-acetylglucosaminyltransferase II (EC 2.4.1.143, GlcNAc-T II) was produced in the Sf9 ***insect*** cell/baculovirus expression system as a fusion protein with a (His)-6 tag and partially purified by affinity chromatography on a metal chelating column. The partially purified enzyme was used to catalyze the transfer of GlcNAc from UDP-GlcNAc to R-alpha-Man(1 fwdarw 6)(beta-GlcNAc(1 fwdarw 2)alpha-Man(1 fwdarw 3))beta-Man-O-octyl to form beta-GlcNAc(1 fwdarw 2)R-alpha-Man(1 fwdarw 6)(beta-GlcNAc(1 fwdarw 2)alpha-Man(1 fwdarw 3))beta-Man-O-octyl where there is either no modification of the alpha-Man(1 fwdarw 6) residue (7), or where R is 3-deoxy (8), 4-deoxy (9) or 6-deoxy (10). The yields ranged from 64-80%. Products were characterized by 1H and 13C nuclear magnetic resonance spectroscopy and fast atom bombardment mass spectrometry. Compounds 7-10 are pentasaccharide analogues of the biantennary N-glycan substrates of N-acetylglucosaminyltransferases III, IV and V.

=> s l1 and cell?
L4 405 L1 AND CELL?

=> s l1 and host cell
L5 11 L1 AND HOST CELL

=> dup rem l5
PROCESSING COMPLETED FOR L5
L6 11 DUP REM L5 (0 DUPLICATES REMOVED)

=> s l6 not l3
L7 6 L6 NOT L3

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 6 ANSWERS - CONTINUE? Y/(N):y

L7 ANSWER 1 OF 6 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2006:1009495 CAPLUS <<LOGINID::20070410>>
DN 145:375282
TI Humanized antibody fragment molecules directed to melanoma chondroitin sulfate proteoglycan and having increased Fc receptor binding affinity and effector function
IN Umana, Pablo; Mossner, Ekkehard
PA Glycart Biotechnology A.-G., Switz.
SO PCT Int. Appl., 187pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2006100582	A1	20060928	WO 2006-IB669	20060324
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
US 2006223096	A1	20061005	US 2006-388204	20060324
PRAI US 2005-665079P	P	20050325		

AB The present invention relates to recombinant monoclonal antibodies that have the binding specificity of murine 225.28S antibody, including chimeric, primatized, or humanized antibodies specific for human melanoma chondroitin sulfate proteoglycan (MCSP, also known as high-mol. wt. melanoma-assocd. antigen or HAA-MAA). In addn., nucleic acid mols. encoding such antigen-binding mols. (ABMs), and vectors and host cells comprising such nucleic acid mols. The invention further relates to methods for producing the ABMs of the invention, and to methods of using these ABMs in treatment of disease. In addn., glycoengineered ABMs are provided with modified glycosylation and having improved therapeutic properties, including antibodies with increased Fc receptor binding (such as Fc.gamma.RIIIa) and increased effector function. Increased effector function can be one or more of increased Fc-mediated cellular cytotoxicity, increased binding to NK cells, increased binding to macrophages, increased binding to monocytes, increased binding to polymorphonuclear cells, direct signaling inducing apoptosis, increased dendritic cell maturation, or increased T cell priming.
RE.CNT 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 2 OF 6 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2005:1293527 CAPLUS <<LOGINID::20070410>>
DN 144:5405
TI Antibody glycosylation variants having increased antibody-dependent cellular cytotoxicity
IN Umana, Pablo; Jean-Mairet, Joel; Bailey, James E.
PA Glycart Biotechnology AG, Switz.
SO U.S. Pat. Appl. Publ., 28 pp., Cont.-in-part of U.S. Ser. No. 633,697.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 3

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2005272128	A1	20051208	US 2005-199232	20050809
US 2003175884	A1	20030918	US 2002-211554	20020805
US 2005079605	A1	20050414	US 2003-633697	20030805
PRAI US 2002-211554	B1	20020805		
US 2003-633697	A2	20030805		
US 1998-82581P	P	19980420		
US 1999-294584	A3	19990420		
US 2001-309516P	P	20010803		

AB The authors disclose glycosylation engineering of host cells to generate proteins with improved therapeutic properties. In one example, therapeutic antibodies expressed by host cells transgenic for N-acetylglucosaminyltransferase exhibit increased antibody-dependent cellular cytotoxicity.

L7 ANSWER 3 OF 6 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2005:447673 CAPLUS <<LOGINID::20070410>>

DN 143:20875
TI Differentially expressed gene profile for diagnosing and treating mental disorders
IN Akil, Huda; Atz, Mary; Bunney, William E., Jr.; Choudary, Prabhakara V.; Evans, Simon J.; Jones, Edward G.; Li, Jun; Lopez, Juan F.; Myers, Richard; Thompson, Robert C.; Tomita, Hiroaki; Vawter, Marquis P.; Watson, Stanley
PA The Board of Trustees of the Leland Stanford Junior University, USA
SO PCT Int. Appl., 226 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2005046434	A2	20050526	WO 2004-US36784	20041105
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2005209181	A1	20050922	US 2004-982556	20041104
AU 2004289247	A1	20050526	AU 2004-289247	20041105
CA 2543811	A1	20050526	CA 2004-2543811	20041105
EP 1680009	A2	20060719	EP 2004-800741	20041105
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, HR, IS, YU				

PRAI US 2003-517751P P 20031105
US 2004-982556 A 20041104
WO 2004-US36784 W 20041105
AB The present invention provides methods for diagnosing mental disorders (e.g., psycholic disorders such as schizophrenia). The present invention uses DNA microarray anal. to demonstrate differential expression of genes in selected regions of post-mortem brains from patients diagnosed with mental disorders in comparison with normal control subjects. The invention also provides methods of identifying modulators of such mental disorders as well as methods of using these modulators to treat patients suffering from such mental disorders.

L7 ANSWER 4 OF 6 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:718660 CAPLUS <<LOGINID::20070410>>
DN 141:237741
TI Production of modified glycoproteins having multiple antennary structures by expression of glucosaminyltransferases in fungal cells
IN Bobrowicz, Piotr; Hamilton, Stephen R.; Gerngross, Tilman U.; Wildt, Stefan; Choi, Byung-Kwon; Nett, Juergen Hermann; Davidson, Robert C.
PA USA
SO PCT Int. Appl., 231 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 25

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2004074461	A2	20040902	WO 2004-US5191	20040220
WO 2004074461	A3	20050317		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2004018590	A1	20040129	US 2003-371877	20030220
US 2005208617	A1	20050922	US 2003-680963	20031007
AU 2004213868	A1	20040902	AU 2004-213868	20040220
CA 2516550	A1	20040902	CA 2004-2516550	20040220
EP 1597381	A2	20051123	EP 2004-713388	20040220
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
JP 2006518600	T	20060817	JP 2006-503776	20040220
US 2007037248	A1	20070215	US 2006-546101	20060803

PRAI US 2003-371877 A 20030220
US 2003-680963 A 20031007
US 2000-214358P P 20000628
US 2000-215638P P 20000630
US 2001-279997P P 20010330
US 2001-892591 A2 20010627
US 2001-344169P P 20011227
WO 2002-US241510 W 20021224
WO 2002-US41510 A2 20021224
WO 2004-US5191 A 20040220
US 2005-500240 A2 20050323
AB The present invention relates to eukaryotic host cells, esp. lower eukaryotic host cells, having modified oligosaccharides which may be modified further by heterologous expression of a set of glycosyltransferases, sugar and sugar nucleotide transporters to become host-strains for the prodn. of mammalian, e.g., human therapeutic glycoproteins. The process provides an engineered ***host*** ***cell*** which can be used to express and target any desirable gene(s) involved in glycosylation. Host cells with modified lipid-linked oligosaccharides are created or selected. N-glycans made in the engineered host cells are substrates for ***GnTIII*** , GnTIV, GnTV, GnT VI or GnTIX activity, which produce bisected and/or multiantennary N-glycan structures and may be modified further by heterologous expression of one or more enzymes, e.g., glycosyltransferases, sugar, sugar nucleotide transporters, to yield human-like glycoproteins. For the prodn. of therapeutic proteins, this method may be adapted to engineer cell lines in which any desired glycosylation structure may be obtained. The invention is illustrated by prodn. of the kringle 3 domain of human plasminogen and interferon-.beta. in engineered Pichia pastoris or Kluyveromyces lactis strains. N-glycans of secreted kringle 3 glycoproteins from Pichia pastoris strains had masses corresponding to GlcNAc1-3Man3-5GlcNAc2.

L7 ANSWER 5 OF 6 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2003:117838 CAPLUS <<LOGINID::20070410>>
DN 138:152272
TI Antibody glycosylation variants having increased antibody-dependent cellular cytotoxicity
IN Jean-Mairet, Joel; Umana, Pablo; Bailey, James E.
PA Glycart Biotechnology AG, Switz.; Bailey, Sean
SO PCT Int. Appl., 68 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 3

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2003011878	A2	20030213	WO 2002-US24739	20020805
WO 2003011878	A3	20031106		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2455365	A1	20030213	CA 2002-2455365	20020805
EP 1423510	A2	20040602	EP 2002-778191	20020805
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK				
CN 1555411	A	20041215	CN 2002-818173	20020805
JP 2005524379	T	20050818	JP 2003-517069	20020805
NO 2004000453	A	20040330	NO 2004-453	20040202
IN 2004KN00195	A	20060407	IN 2004-KN195	20040213

PRAI US 2001-309516P P 20010803
WO 2002-US24739 W 20020805
AB The authors disclose glycosylation engineering of antibodies. In one example, the antibody-dependent cellular cytotoxicity of the therapeutic antibody IDEC-C2B8 was shown to be enhanced by the increased glycosylation derived from its prodn. by CHO cells transfected for expression of .beta.(1,4)- ***N*** - ***acetylglucosaminyltransferase*** ***III*** .

L7 ANSWER 6 OF 6 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1999:691109 CAPLUS <<LOGINID::20070410>>
DN 131:335805
TI Glycosylation engineering of antibodies for improving antibody-dependent cellular cytotoxicity
IN Umana, Pablo; Jean-Mairet, Joel; Bailey, James E.
PA Switz.
SO PCT Int. Appl., 79 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 3

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9954342	A1	19991028	WO 1999-US8711	19990420
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 9936578	A	19991108	AU 1999-36578	19990420
EP 1071700	A1	20010131	EP 1999-918731	19990420
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
JP 2002512014	T	20020423	JP 2000-544680	19990420
US 6602684	B1	20030805	US 1999-294584	19990420
US 2004072290	A1	20040415	US 2003-437388	20030514

US 2005074843 A1 20050407 US 2003-633699 20030805
PRAI US 1998-82581P P 19980420
US 1999-294584 A1 19990420
WO 1999-US8711 W 19990420
AB The present invention relates to the field of glycosylation engineering of proteins. More particularly, the present invention is directed to the glycosylation engineering of proteins to provide proteins with improved therapeutic properties, e.g., antibodies, antibody fragments, or a fusion protein that includes a region equiv. to the Fc region of an Ig, with enhanced Fc-mediated cellular cytotoxicity. The antibodies or fusion proteins with enhanced Fc-mediated cellular cytotoxicity are expressed in host cells engineered to also express a glycoprotein-modifying glycosyl transferase, e.g. .beta.(1,4)- ***N*** - ***acetylglucosaminyltransfera*** *** se*** ***III*** or V, .beta.(1,4)-N-galactosyltransferase, and mannosidase II.
RE.CNT 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d his

(FILE 'HOME' ENTERED AT 14:34:32 ON 10 APR 2007)

FILE 'EMBASE, BIOSIS, CAPLUS' ENTERED AT 14:34:44 ON 10 APR 2007
L1 510 S N ACETYLGLUCOSAMINYLTRANSFERASE III OR GNTIII OR GNT III
L2 22 S L1 AND (YEAST OR FUNGI OR INSECT OR PLANT)
L3 17 DUP REM L2 (5 DUPLICATES REMOVED)
L4 405 S L1 AND CELL?
L5 11 S L1 AND HOST CELL
L6 11 DUP REM L5 (0 DUPLICATES REMOVED)
L7 6 S L6 NOT L3

=> s N acetylglucosaminyltransferase II or GnTII or GnT II
L8 236 N ACETYLGLUCOSAMINYLTRANSFERASE II OR GNTII OR GNT II

=> s l1 and l8
L9 18 L1 AND L8

=> dup rem l9
PROCESSING COMPLETED FOR L9
L10 16 DUP REM L9 (2 DUPLICATES REMOVED)

=> s l10 not l3
L11 7 L10 NOT L3

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 7 ANSWERS - CONTINUE? Y/(N):y

L11 ANSWER 1 OF 7 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2005:280390 BIOSIS <<LOGINID::20070410>>
DN PREV200510065099
TI The relationship between the branch-forming glycosyltransferases and cell surface sugar chain structures.
AU Takamatsu, Shinji [Reprint Author]; Inoue, Noboru; Katsumata, Toshiyuki; Nakamura, Katsumi; Fujibayashi, Yasuhisa; Takeuchi, Makoto
CS Fukui Univ, Biomed Imaging Res Ctr, 23-3 Shimoaizuki, Fukui 9101193, Japan
shinjit@fmsrsa.fukui-med.ac.jp
SO Biochemistry, (APR 26 2005) Vol. 44, No. 16, pp. 6343-6349.
CODEN: BICHAW. ISSN: 0006-2960.
DT Article
LA English
ED Entered STN: 27 Jul 2005
Last Updated on STN: 27 Jul 2005
AB Many recombinant proteins developed or under development for clinical use are glycoproteins, and trials aimed at improving their bioactivity or pharmacokinetics in vivo by altering specific glycan structures are ongoing. For pharmaceuticals of glycoproteins, it is important to characterize and, if possible, control the glycosylation profile. However, the mechanism responsible for the regulation of sugar chain structures found on naturally occurring glycoproteins is still unclear. To clarify the relationship between glycosyltransferases and sugar chain branch structure, we estimated six glycosyltransferases' activities (N-acetylglucosaminyltransferase (GlcNAcTase)-I, -II, -III, -IV, -V, and beta-1,4-galactosyltransferase (GalT)) which control the branch formation on asparagine (Asn)-linked sugar chains in 18 human cancer cell lines derived from several tissues. To visualize the balance of glycosyltransferase activity associated with each cell line, we expressed the relative glycosyltransferase activity in comparison to the average activity among the cell lines. These cell lines were classified into five groups according to their relative glycosyltransferase balance and were termed GlcNAcTase-I/II, GlcNAcTase-III, GlcNAcTase-IV, GlcNAcTase-V, and GalT. We also characterized the structures of Asn-linked sugar chains on the cell surface of representative cell lines of each group. The branching structure of cell surface sugar chains roughly corresponded to the glycosyltransferase balance. This finding suggests that, for the sugar chain structure remodeling of glycoproteins, attention should be focused on the glycosyltransferase balance of host cells before introducing exogenous glycosyltransferases or down-regulating the activity of intrinsic glycosyltransferases.

L11 ANSWER 2 OF 7 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2002:199717 BIOSIS <<LOGINID::20070410>>
DN PREV200200199717
TI Cloning and expression of a novel UDP-GlcNAc:alpha-D-mannoside beta1,2-N-acetylglucosaminyltransferase homologous to UDP-GlcNAc:alpha-3-D-
mannoside beta1,2-N-acetylglucosaminyltransferase I.
AU Zhang, Wenli; Betel, Doron; Schachter, Harry [Reprint author]
CS Program in Structural Biology and Biochemistry, The Research Institute, Hospital for Sick Children, 555 University Avenue, Toronto, Ontario, M5G 1X8, Canada
harry@sickkids.on.ca
SO Biochemical Journal, (1 January, 2002) Vol. 361, No. 1, pp. 153-162.
print.
ISSN: 0264-6021.
DT Article
LA English
ED Entered STN: 20 Mar 2002
Last Updated on STN: 20 Mar 2002
AB A TBLASTN search with human UDP-GlcNAc:alpha-3-mannoside beta-1,2-N-acetylglucosaminyltransferase I (GnT I; EC 2.4.1.101) as a probe identified human and mouse Unigenes encoding a protein similar to human GnT I (34% identity over 340 amino acids). The recombinant protein converted Man(alpha1-6)(Man(alpha1-3))Man(beta1-)O-octyl to Man(alpha1-6)(GlcNAc(beta1-2)Man(alpha1-3)) Man(beta1-)O-octyl, the reaction catalysed by GnT I. The enzyme also added GlcNAc to Man(alpha1-6)(GlcNAc(beta1-2)Man(alpha1-3)) Man(beta1-)O-octyl (the substrate for beta-1,2- ***N*** - ***acetylglucosaminyltransferase*** ***II***), Man(alpha1-)O-benzyl (with Km values of apprxeq 0.3 and > 30 mM for UDP-GlcNAc and Man(alpha1-)O-benzyl respectively) and the glycopeptide CYA(Man(alpha1-)O-T)AV (Km apprx 12 mM). The product formed
with Man(alpha1-)O-benzyl was identified as GlcNAc(beta1-2)Man(alpha1-)O-benzyl by proton NMR spectroscopy. The enzyme was named UDP-GlcNAc:alpha-D-mannoside beta-1,2-N-acetylglucosaminyltransferase I.2 (GnT I.2). The human gene mapped to chromosome 1. Northern-blot analysis showed a 3.3 kb message with a wide tissue distribution. The cDNA has a 1980 bp open reading frame encoding a 660 amino acid protein with a type-2 domain structure typical of glycosyltransferases. Man(beta1-)O-octyl, Man(beta1-)O-p-nitrophenyl and GlcNAc(beta1-2)Man(alpha1-6)(GlcNAc(beta1-2)Man(alpha1-3))Man(beta1-4)GlcNAc(beta1-4)GlcNAc(beta1-)O-Asn were not acceptors, indicating that GnT I.2 is specific for alpha-linked terminal Man and does not have ***N*** - ***acetylglucosaminyltransferase*** ***III*** , IV, V, VII or VIII activities. CYA(Man(alpha1-)O-T)AV was between three and seven times more effective as an acceptor than the other substrates, suggesting that GnT I.2 may be responsible for the synthesis of the GlcNAc(beta1-2)Man(alpha1-)O-Ser/Thr moiety on alpha-dystroglycan and other O-mannosylated proteins.

L11 ANSWER 3 OF 7 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 1987:315149 BIOSIS <<LOGINID::20070410>>
DN PREV198784034656; BA84:34656
TI CONTROL OF GLYCOPROTEIN SYNTHESIS KINETIC MECHANISM SUBSTRATE SPECIFICITY
AND INHIBITION CHARACTERISTICS OF UDP-N ACETYLGLUCOSAMINE ALPHA-D
MANNOSIDE BETA-1-2 ***N***
ACETYLGLUCOSAMINYLTRANSFERASE
II FROM RAT LIVER.
AU BENDIAK B [Reprint author]; SCHACHTER H
CS BIOCHEM DEP, RES INST HOSP SICK CHILDREN, UNIV TORONTO, TORONTO, ONTARIO
M5G 1X8, CANADA
SO Journal of Biological Chemistry, (1987) Vol. 262, No. 12, pp. 5784-5790.
CODEN: JBCHA3. ISSN: 0021-9258.
DT Article
FS BA
LA ENGLISH
ED Entered STN: 25 Jul 1987
Last Updated on STN: 25 Jul 1987
AB Purified rat liver UDP-GlcNAc:.alpha.-D-mannoside .beta.1-2 ***N*** - ***acetylglucosaminyltransferase*** ***II*** (Bendiak, B., and Schachter, H. (1987) J. Biol. Chem. 262, 5775-5783) has been characterized kinetically, and its substrate specificity and inhibition characteristics have been determined. Kinetic data indicate an ordered, or largely ordered sequential mechanism, with UDP-GlcNAc binding prior to the acceptor. The minimal acceptor structure required for full activity was determined. The acceptor molecule must have a terminal Man.alpha.1-6 residue, and a terminal GlcNAc.beta.1-2Man.alpha.1-3 branch to display any activity, but does not require the reducing GlcNAc residue, as the enzyme was about 50% as active after reduction of this residue to N-acetylglucoasaminitol. Additional residues (Gal.beta.1-4 on the GlcNAc.beta.1-2Man.alpha.1-3 arm, or a bisecting GlcNAc.beta.1-4 on the .beta.-Man residue) abolish catalytic activity. These results suggest a rigid order in the biosynthesis of all N-linked complex oligosaccharides (bisected and nonbisected bi-, tri-, and tetraantennary), since the enzyme must act to completion prior to the action of either UDP-Gal:GlcNAc .beta.1-4 galactosyltransferase or ***N*** - ***acetylglucosaminyltransferase*** ***III*** to make such structures. Inhibition studies with nucleotides, sugars,

nucleotide-sugars, and their respective analogues revealed that analogues of UDP and UTP, in which the hydrogen at the 5 position of the uracil was substituted with -CH3, bromine, or mercury (as the mercaptide) were good reversible inhibitors of the enzyme, whereas substitution at other sites lessened the inhibitory potency, usually to a large degree.

L11 ANSWER 4 OF 7 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2005:429444 CAPLUS <<LOGINID::20070410>>
DN 142:480781
TI Human CD20-specific humanized, chimeric and primatized antibodies with modified glycosylation to increase antigen/Fc receptor binding and effector function for treating B cell lymphoma
IN Umana, Pablo; Bruenker, Peter; Ferrara, Claudia; Suter, Tobias; Puentener, Ursula; Moessner, Ekkehard
PA Glycart Biotechnology A.-G., Switz.
SO PCT Int. Appl., 187 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2005044859	A2	20050519	WO 2004-IB3896	20041105
WO 2005044859	A3	20050804		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2004287643	A1	20050519	AU 2004-287643	20041105
CA 2544865	A1	20050519	CA 2004-2544865	20041105
US 2005123546	A1	20050609	US 2004-981738	20041105
EP 1692182	A2	20060823	EP 2004-798998	20041105
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, HR, IS				
BR 2004016262	A	20070109	BR 2004-16262	20041105
CN 1902231	A	20070124	CN 2004-80039946	20041105
NO 2006002289	A	20060803	NO 2006-2289	20060519
PRAI US 2003-517096P	P	20031105		
WO 2004-IB3896	W	20041105		

AB The present invention relates to antigen binding mols. (ABMs). In particular embodiments, the present invention relates to recombinant monoclonal antibodies, including chimeric, primatized or humanized antibodies specific for human CD20. In addn., the present invention relates to nucleic acid mols. encoding such ABMs, and vectors and host cells comprising such nucleic acid mols. The invention further relates to methods for producing the ABMs of the invention, and to methods of using these ABMs in treatment of disease. In addn., the present invention relates to ABMs with modified glycosylation having improved therapeutic properties, including antibodies with increased Fc receptor binding and increased effector function.

L11 ANSWER 5 OF 7 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:718660 CAPLUS <<LOGINID::20070410>>
DN 141:237741
TI Production of modified glycoproteins having multiple antennary structures by expression of glucosaminyltransferases in fungal cells
IN Bobrowicz, Piotr; Hamilton, Stephen R.; Gerngross, Tilman U.; Wildt, Stefan; Choi, Byung-Kwon; Nett, Juergen Hermann; Davidson, Robert C.
PA USA
SO PCT Int. Appl., 231 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 25

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2004074461	A2	20040902	WO.2004-US5191	20040220
WO 2004074461	A3	20050317		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2004018590	A1	20040129	US 2003-371877	20030220
US 2005208617	A1	20050922	US 2003-680963	20031007
AU 2004213868	A1	20040902	AU 2004-213868	20040220
CA 2516550	A1	20040902	CA 2004-2516550	20040220
EP 1597381	A2	20051123	EP 2004-713388	20040220
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
JP 2006518600	T	20060817	JP 2006-503776	20040220
US 2007037248	A1	20070215	US 2006-546101	20060803
PRAI US 2003-371877	A	20030220		
US 2003-680963	A	20031007		
US 2000-214358P	P	20000628		
US 2000-215638P	P	20000630		
US 2001-279997P	P	20010330		
US 2001-892591	A2	20010627		
US 2001-344169P	P	20011227		
WO 2002-US241510	W	20021224		
WO 2002-US41510	A2	20021224		
WO 2004-US5191	A	20040220		
US 2005-500240	A2	20050323		

AB The present invention relates to eukaryotic host cells, esp. lower eukaryotic host cells, having modified oligosaccharides which may be modified further by heterologous expression of a set of glycosyltransferases, sugar and sugar nucleotide transporters to become host-strains for the prodn. of mammalian, e.g., human therapeutic glycoproteins. The process provides an engineered host cell which can be used to express and target any desirable gene(s) involved in glycosylation. Host cells with modified lipid-linked oligosaccharides are created or selected. N-glycans made in the engineered host cells are substrates for ***GnTIII*** , GnTIV, GnTV, GnT VI or GnTIX activity, which produce bisected and/or multiantennary N-glycan structures and may be modified further by heterologous expression of one or more enzymes, e.g., glycosyltransferases, sugar, sugar nucleotide transporters, to yield human-like glycoproteins. For the prodn. of therapeutic proteins, this method may be adapted to engineer cell lines in which any desired glycosylation structure may be obtained. The invention is illustrated by prodn. of the kringle 3 domain of human plasminogen and interferon-.beta. in engineered Pichia pastoris or Kluyveromyces lactis strains. N-glycans of secreted kringle 3 glycoproteins from Pichia pastoris strains had masses corresponding to GlcNAc1-3Man3-5GlcNAc2.

L11 ANSWER 6 OF 7 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2000:716357 CAPLUS <<LOGINID::20070410>>
DN 134:96939
TI Branching of N-glycans: N-acetylglucosaminyltransferases
AU Schachter, Harry
CS Department of Biochemistry Hospital for Sick Children, Toronto, ON, M5G 1X8, Can.
SO Carbohydrates in Chemistry and Biology (2000), Volume 3, 145-173. Editor(s): Ernst, Beat; Hart, Gerald W.; Sinay, Pierre. Publisher: Wiley-VCH Verlag GmbH, Weinheim, Germany.
CODEN: 69AMJE
DT Conference; General Review
LA English
AB A review with 221 refs. is presented regarding N-acetylglucosaminyltransferases which initiate the branches of complex N-glycans. Topics discussed include the processing of N-glycans within the endomembrane assembly line, the general properties of N-acetylglucosaminyltransferases (GnTs), the role of GnT I and ***GnT*** ***II*** in mammalian development, and the roles of ***GnT*** ***III*** , GnT IV, GnT V, GnT VI, GnT VII and GnT VIII.
RE.CNT 222 THERE ARE 222 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 7 OF 7 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1988:418572 CAPLUS <<LOGINID::20070410>>
DN 109:18572
TI Expression of ***N*** - ***acetylglucosaminyltransferase*** ***III*** in hepatic nodules during rat liver carcinogenesis promoted by orotic acid
AU Narasimhan, Saroja; Schachter, Harry; Rajalakshmi, Srinivasan
CS Dep. Biochem. Res., Hosp. Sick Child., Toronto, ON, M5G 1X8, Can.
SO Journal of Biological Chemistry (1988), 263(3), 1273-81
CODEN: JBCHA3; ISSN: 0021-9258
DT Journal
LA English
GI

/ Structure 1 in file .gra /

AB The activity of ***N*** - ***acetylglucosaminyltransferase*** ***III*** was detd. in hepatic nodules of rats initiated by administration of a single dose of the carcinogen 1,2-dimethylhydrazine.2HCl (100 mg/kg, i.p.) 18 h after partial hepatectomy and promoted by feeding a diet supplemented with 1% orotic acid (I) for 32-40 wk. The nodules had significant ***N*** - ***acetylglucosaminyltransferase*** ***III*** activity (0.78-2.18 nmol GlcNAc transferred/h/mg of protein), whereas the surrounding liver, the regenerating liver (24 h after partial hepatectomy), and the control liver had negligible activity (0.02-0.03 nmol/h/mg of protein). Product isolated from a large scale ***N*** - ***acetylglucosaminyltransferase*** *** *** ***III*** incubation with hepatic nodules as enzyme source showed the presence of the bisecting GlcNAc residue by 500 MHz proton NMR spectroscopy. Concomitant with the appearance of ***N*** - ***acetylglucosaminyltransferase*** ***III*** activity in the preneoplastic nodules, the activities of N-acetylglucosaminyltransferase I and II were decreased in these membranes when compared to those from surrounding liver, regenerating liver, and control liver. Apparently, ***N*** - ***acetylglucosaminyltransferase*** ***III*** is induced

at the preneoplastic stage in liver carcinogenesis promoted by I and are consistent with the reported presence of bisecting GlcNAc residues in the Asn-linked oligosaccharides of rat and human hepatoma .gamma.-glutamyl transpeptidase and their absence in enzyme from normal liver of rats and humans (A. Kobata, and K. Yamashita, (1984)].

=> d his

(FILE 'HOME' ENTERED AT 14:34:32 ON 10 APR 2007)

FILE 'EMBASE, BIOSIS, CAPLUS' ENTERED AT 14:34:44 ON 10 APR 2007
L1 510 S N ACETYLGLUCOSAMINYLTRANSFERASE III OR GNTIII OR GNT III
L2 22 S L1 AND (YEAST OR FUNGI OR INSECT OR PLANT)
L3 17 DUP REM L2 (5 DUPLICATES REMOVED)
L4 405 S L1 AND CELL?
L5 11 S L1 AND HOST CELL
L6 11 DUP REM L5 (0 DUPLICATES REMOVED)
L7 6 S L6 NOT L3
L8 236 S N ACETYLGLUCOSAMINYLTRANSFERASE II OR GNTII OR GNT II
L9 18 S L1 AND L8
L10 16 DUP REM L9 (2 DUPLICATES REMOVED)
L11 7 S L10 NOT L3

=> s l1 and py<=2003
L12 395 L1 AND PY<=2003

=> s l12 and N glycan
L13 34 L12 AND N GLYCAN

=> dup rem l13
PROCESSING COMPLETED FOR L13
L14 19 DUP REM L13 (15 DUPLICATES REMOVED)

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 19 ANSWERS - CONTINUE? Y/(N):y

L14 ANSWER 1 OF 19 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2007:173778 CAPLUS <<LOGINID::20070410>>
DN 146:227824
TI Transgenic eukaryotic microorganisms expressing mammalian genes for enzymes of protein glycosidation and their use in the manufacture of proteins with multiple antennary carbohydrate structures
IN Bobrowicz, Piotr; Hamilton, Stephen R.; Gerngross, Tillman U.; Wildt, Stefan; Choi, Byung-Kwon; Nett, Juergen Hermann; Davidson, Robert C.
PA USA
SO U.S. Pat. Appl. Publ., 183pp., Cont.-in-part of U.S. Ser. No. 500,240.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 25

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 2007037248	A1	20070215	US 2006-546101	20060803
	EP 1522590	A1	20050413	EP 2004-25648	20010627
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY, TR				
	WO 2003056914	A1	20030717	WO 2002-US41510	20021224 <--
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	US 2004018590	A1	20040129	US 2003-371877	20030220
	US 2005208617	A1	20050922	US 2003-680963	20031007
	WO 2004074461	A2	20040902	WO 2004-US5191	20040220
	WO 2004074461	A3	20050317		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI				
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PRAI	US 2000-214358P	P	20000628		
	US 2000-215638P	P	20000630		
	US 2001-279997P	P	20010330		
	US 2001-344169P	P	20011227		
	WO 2002-US241510	W	20021224		
	US 2003-371877	A	20030220		
	US 2003-680963	A	20031007		
	WO 2004-US5191	W	20040220		
	US 2005-500240	A2	20050323		
	EP 2001-954606	A3	20010627		
	US 2001-892591	A2	20010627		
	WO 2002-US41510	A2	20021224		

AB The present invention relates to eukaryotic host cells, esp. lower eukaryotic host cells, capable of manufg. glycoproteins with mammalian glycosidation structures for therapeutic use. These cells express mammalian genes for enzymes involved in the synthesis of precursors for glycosylation and their incorporation into antennary carbohydrate structures. N-glycans made in these host cells exhibit ***GnTIII*** , GnTIV, GnTV, GnT VI or GnTIX activity, which produce bisected and/or multiantennary ***N*** - ***glycan*** structures and may be modified further by heterologous expression of one or more enzymes, e.g., glycosyltransferases, sugar, sugar nucleotide transporters, to yield human-like glycoproteins. For the prodn. of therapeutic proteins, this method may be adapted to engineer cell lines in which any desired glycosylation structure may be obtained.

L14 ANSWER 2 OF 19 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2005:1028015 CAPLUS <<LOGINID::20070410>>
DN 143:300313
TI N-acetylglucosamintransferase III expression in genetically modified lower eukaryotes
IN Bobrowicz, Piotr; Hamilton, Stephen R.; Gerngross, Tilman U.; Wildt, Stefan; Choi, Byung-Kwon; Nett, Juergen Hermann; Davidson, Robert C.
PA USA
SO U.S. Pat. Appl. Publ., 163 pp., Cont.-in-part of U.S. Ser. No. 371,877.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 25

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 2005208617	A1	20050922	US 2003-680963	20031007
	US 2002137134	A1	20020926	US 2001-892591	20010627 <--
	US 7029872	B2	20060418		
	EP 1522590	A1	20050413	EP 2004-25648	20010627
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY, TR				
	WO 2003056914	A1	20030717	WO 2002-US41510	20021224 <--
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	US 2004018590	A1	20040129	US 2003-371877	20030220
	AU 2004213859	A1	20040902	AU 2004-213859	20040220
	AU 2004213868	A1	20040902	AU 2004-213868	20040220
	CA 2516520	A1	20040902	CA 2004-2516520	20040220
	CA 2516550	A1	20040902	CA 2004-2516550	20040220
	WO 2004074458	A2	20040902	WO 2004-US5128	20040220
	WO 2004074458	A3	20041229		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI				
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	WO 2004074461	A2	20040902	WO 2004-US5191	20040220
	WO 2004074461	A3	20050317		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI				
	RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	EP 1597381	A2	20051123	EP 2004-713388	20040220
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
	EP 1599595	A2	20051130	EP 2004-713412	20040220
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
	JP 2006518597	T	20060817	JP 2006-503757	20040220
	JP 2006518600	T	20060817	JP 2006-503776	20040220
	US 2007037248	A1	20070215	US 2006-546101	20060803
PRAI	US 2000-214358P	P	20000628		
	US 2000-215638P	P	20000630		
	US 2001-279997P	P	20010330		
	US 2001-892591	A2	20010627		
	US 2001-344169P	P	20011227		
	WO 2002-US41510	A2	20021224		
	US 2003-371877	A2	20030220		
	EP 2001-954606	A3	20010627		
	WO 2002-US241510	W	20021224		
	US 2003-680963	A	20031007		
	WO 2004-US5128	A	20040220		
	WO 2004-US5191	A	20040220		

US 2005-500240 A2 20050323

AB The present invention relates to eukaryotic host cells having modified oligosaccharides which may be modified further by heterologous expression of a set of glycosyltransferases, sugar transporters and mannosidases to become host-strains for the prodn. of mammalian, e.g., human therapeutic glycoproteins. The present invention relates to methods and comps. by which non-human eucaryotic cells, such as fungi or other eukaryotic cells, can be genetically modified to produce glycosylated proteins (glycoproteins) having patterns of glycosylation similar to those of glycoproteins produced by animal cells, esp. human cells, which are useful as human or animal therapeutic agents. The process provides an engineered host cell which can be used to express and target any desirable gene(s) involved in glycosylation. Host cells with modified lipid-linked oligosaccharides are created or selected. N-glycans made in the engineered host cells exhibit ***GnTIII*** activity, which produce bisected ***N*** - ***glycan*** structures and may be modified further by heterologous expression of one or more enzymes, e.g., glycosyltransferases, sugar transporters and mannosidases, to yield human-like glycoproteins. For the prodn. of therapeutic proteins, this method may be adapted to engineer cell lines in which any desired glycosylation structure may be obtained.

L14 ANSWER 3 OF 19 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2003:757842 CAPLUS <<LOGINID::20070410>>
DN 139:272047
TI Mammalian UDP-N-acetylglucosamine:.beta.-D-mannoside .beta.(1,4)-N-acetylglucosaminyltransferase (***GnTIII***) expression in plants
IN Bakker, Hendrikus Antonius Cornelus; Florack, Dionisius Elisabeth Antonius; Bosch, Hendrik Jan
PA Plant Research International B.V., Neth.
SO PCT Int. Appl., 122 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2003078614	A2	20030925	WO 2003-IB1562	20030318 <--
WO 2003078614	A3	20040108		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2478294	A1	20030925	CA 2003-2478294	20030318 <--
AU 2003219402	A1	20030929	AU 2003-219402	20030318 <--
EP 1485492	A2	20041215	EP 2003-715213	20030318
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
CN 1665934	A	20050907	CN 2003-806546	20030318
JP 2005528092	T	20050922	JP 2003-576608	20030318
NZ 534881	A	20060929	NZ 2003-534881	20030318
US 2005223430	A1	20051006	US 2004-508166	20040917
PRAI US 2002-365769P	P	20020319		
US 2002-368047P	P	20020326		
US 2002-365769	P	20020319		
US 2002-368047	P	20020326		
WO 2003-IB1562	W	20030318		

AB The invention relates to the field of glycoprotein processing in transgenic plants used as cost efficient and contamination safe factories for the prodn. of recombinant biopharmaceutical proteins or pharmaceutical comps. comprising these glycoproteins. The invention provides a plant comprising a functional mammalian enzyme providing mammalian UDP-N-acetylglucosamine:.beta.-D-mannoside .beta.(1,4)-N-acetylglucosaminyltransferase (***GnTIII***) that is normally not present in plants, said plant addnl. comprising at least a second mammalian protein or functional fragment thereof that is normally not present in plants. The invention further relates to a hybrid protein comprising the catalytic site of ***GnTIII*** and transmembrane domain of Golgi app. and/or endoplasmic reticulum (ER) protein or modified ***GnTIII*** comprising ER retention signals and its use in producing glycoproteins with oligosaccharides that lack immunogenic xylose and fucose residues. Thus, the human gene for ***GnTIII*** was cloned, and a C-terminal c-myc tag for anal. of expression of the tagged fusion protein was provided and the whole was placed under the control of plant regulatory elements for introduction in maize. ***GnTIII*** is expressed in plants and expression results in bisected oligosaccharide structures on endogenous plant glycoproteins. The amt. of N-glycans contg. at lease two GlcNAc residues more than doubled compared to those found in normal maize plants. Expression of ***GnTIII*** also resulted in a significant redn. of complex type ***N*** - ***glycan*** degrdn. products as apparent from matrix-assisted laser desorption ionization time-of-flight (MALDI-TOF) analyses of the isolated glycans of endogenous plant glycoproteins. These data suggest that expression of ***GnTIII*** in maize resulting in the introduction of bisected structures on N-glycans protects the glycans from degrdn. by .beta.-N-acetylhexosaminidases.

L14 ANSWER 4 OF 19 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2003:551280 CAPLUS <<LOGINID::20070410>>
DN 139:112733
TI Methods for production of recombinant glycoproteins with mammalian-type carbohydrate structures and their use for production of immunoglobulins
IN Wildt, Stefan; Miele, Robert Gordon; Nett, Juergen Hermann; Davidson, Robert C.
PA Glycofi, Inc., USA
SO PCT Int. Appl., 125 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 25

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2003056914	A1	20030717	WO 2002-US41510	20021224 <--
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2471551	A1	20030717	CA 2002-2471551	20021224 <--
AU 2002358296	A1	20030724	AU 2002-358296	20021224 <--
EP 1467615	A1	20041020	EP 2002-792535	20021224
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK				
JP 2005514021	T	20050519	JP 2003-557288	20021224
US 2005170452	A1	20050804	US 2003-500240	20021224
US 2004230042	A1	20041118	US 2003-616082	20030708
US 2005208617	A1	20050922	US 2003-680963	20031007
US 2006040353	A1	20060223	US 2005-108088	20050415
US 2006024292	A1	20060202	US 2005-187065	20050721
US 2006029604	A1	20060209	US 2005-187229	20050721
US 2006034829	A1	20060216	US 2005-187079	20050721
US 2006034830	A1	20060216	US 2005-187113	20050721
US 2006286637	A1	20061221	US 2006-429672	20060505
US 2007037248	A1	20070215	US 2006-546101	20060803
PRAI US 2001-344169P	P	20011227		
US 2000-214358P	P	20000628		
US 2000-215638P	P	20000630		
US 2001-279997P	P	20010330		
US 2001-892591	A2	20010627		
WO 2002-US241510	W	20021224		
WO 2002-US41510	W	20021224		
US 2003-371877	A2	20030220		
US 2003-680963	A	20031007		
WO 2004-US5191	W	20040220		
US 2004-554139P	P	20040317		
US 2004-562424P	P	20040415		
US 2004-589913P	P	20040721		
US 2004-589937P	P	20040721		
US 2004-590011P	P	20040721		
US 2004-590030P	P	20040721		
US 2004-590051P	P	20040721		
US 2004-590052P	P	20040721		
US 2004-639657P	P	20041223		
US 2004-639698P	P	20041223		
US 2005-84624	A2	20050317		
US 2005-500240	A2	20050323		
US 2005-108088	A2	20050415		

AB The present invention relates to host cells having modified lipid-linked oligosaccharides which may be modified further by heterologous expression of a set of glycosyltransferases, sugar transporters and mannosidases to become host-strains for the prodn. of mammalian, e.g., human therapeutic glycoproteins. The process provides an engineered host cell which can be used to express and target any desirable gene(s) involved in glycosylation. Host cells with modified lipid-linked oligosaccharides are created or selected. N-glycans made in the engineered host cells have a GlcNAcMan3GlcNAc2 core structure which may then be modified further by heterologous expression of one or more enzymes, e.g., glycosyltransferases, sugar transporters and mannosidases, to yield human-like glycoproteins. For the prodn. of therapeutic proteins, this method may be adapted to engineer cell lines in which any desired glycosylation structure may be obtained. The invention specifically claims use of nucleic acid sequences for gene ALG3 from Pichia pastoris. The ALG3 gene encodes the enzyme which transfers a mannose residue to the Man5-GlcNac2-PP-Dol precursor. The invention also claims use of genetically engineered host cells for recombinant prodn. of Igs. In examples of the invention, a Pichia pastoris strain with deletions of genes alg3 and och1 was constructed. This strain was transformed with the Kringle 3 domain of human plasminogen as a glycosylation substrate. Mass spectrometric anal. of N-glycans isolated from the kringle 3 glycoproteins showed GlcNAcMan3GlcNAc2 and GlcNAcMan4GlcNAc2 structures which could be further modified in vitro. Addn. of N-acetylglucosamine to GlcNAcMan3GlcNAc2 by N-acetylglucosaminyltransferases II and III yields a "bisected" ***N*** - ***glycan*** , GlcNAc3Man3GlcNAc2, which has been implicated in greater antibody-dependent cellular cytotoxicity.

Methods of the invention can be used to engineer a yeast strain capable of producing glycoproteins with bisected N-glycans and expressing Ig mols. with bisected N-glycans attached to asparagine residue 297 in the CH2 portion.
RE.CNT 15 THERE ARE 15 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 5 OF 19 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2004:356559 BIOSIS <<LOGINID::20070410>>
DN PREV200400361822
TI Alteration in N-acetylglucosaminyltransferase activities and glycan structure in tissue and bile glycoproteins from extrahepatic bile duct carcinoma.
AU Jin, Xiao-ling; Liu, Hou-bao; Zhang, Ying; Wang, Bing-Sheng; Chen, Hui-li [Reprint Author]
CS Shanghai Med CollDept BiochemMinist Hlth,Key Lab Glycoconjugate Res, Fudan
Univ, 138 Yi Xue Yuan Rd, Shanghai, 200032, China
hlchen@shmu.edu.cn
SO Glycoconjugate Journal, (***2003***) Vol. 20, No. 6, pp. 399-406. print.
ISSN: 0282-0080 (ISSN print).
DT Article
LA English
ED Entered STN: 5 Sep 2004
Last Updated on STN: 5 Sep 2004
AB The activities of three N-acetylglucosaminyltransferases (***GnT***)- ***III*** , IV and V, as well as the structural alterations of N-glycans on the glycoproteins in cancer tissues and bile specimens from 28 cases of extrahepatic bile duct carcinoma (EBDC) were compared with those from 18 cases of benign biliary duct diseases (BBDD). GnT activities were determined with fluorescence-labeled substrate using a HPLC method. It was found that ***GnT*** - ***III*** and GnT-V activities in EBDC were increased to 3.14 and 15.96 times respectively of the mean BBDD values, but GnT-IV remained unchanged. The activity of GnT-V was correlated with the grade of differentiation and TMN stage of EBDC. The up-regulation of ***GnT*** - ***III*** resulted in the increased bisecting-GlcNAc on the N-glycans of glycoproteins in cancer tissues and a 201 kDa bile glycoprotein when analyzed with HRP-labeled E4-PHA. The increased GnT-V activity led to the elevation of the beta1,6GlcNAc branch (or antennary number) on the N-glycans in cancer tissue glycoproteins and 201, 163, 122 kDa proteins in the bile as probed with HRP-labeled DSA. These findings suggest that the alteration in GnT activities may be involved in the malignant transformation and development of EBDC, resulting in the aberrant glycosylation of some tissue and bile proteins. The latter was expected to be used in the clinical diagnosis and prognosis evaluation in EBDC patients. Published in 2004.

L14 ANSWER 6 OF 19 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2004:147696 BIOSIS <<LOGINID::20070410>>
DN PREV200400151172
TI Remodeling of the major mouse xenoantigen, Galalpha1-3Galbeta1-4GlcNAc-R,
by ***N*** - ***acetylglucosaminyltransferase*** - ***III*** .
AU Chung, Tae-Wook; Kim, Kyung-Sook; Kang, Sung-Koo; Lee, Jung-Woong; Song,
Eun-Young; Chung, Tae-Hwa; Yeom, Young-Il; Kim, Cheorl-Ho [Reprint Author]
CS National Research Laboratory for Glycobiology, Department of Biochemistry and Molecular Biology, COM, Korean Ministry of Science and Technology, Dongguk University, Kyungju, 780-714, South Korea
chkimbio@dongguk.ac.kr
SO Molecules and Cells, (***December 31 2003***) Vol. 16, No. 3, pp. 343-353. print.
ISSN: 1016-8478 (ISSN print).
DT Article
LA English
ED Entered STN: 17 Mar 2004
Last Updated on STN: 17 Mar 2004
AB beta-D-Mannoside beta-1,4- ***N*** - ***acetylglucosaminyltransferase***** *** * ***III*** (***GnT*** - ***III***) catalyses the attachment of an N-acetylglucosamine (GlcNAc) residue to mannose in the beta(1-4) configuration in N-glycans, and forms a bisecting GlcNAc. We have generated transgenic mice that contain the human ***GnT*** - ***III*** gene under the control of the mouse albumin enhancer/promoter (Lee et al., (2003)). Overexpression of this gene in mice reduced the antigenicity of N-glycans to human natural antibodies, especially in the case of the alpha-Gal epitope, Galalpha1-3Galbeta1-4GlcNAc-R. Study of endothelial cells from the ***GnT*** - ***III*** transgenic mice revealed a significant reduction in antigenicity, and a dramatic decrease in both complement- and natural killer cell-mediated mouse cell lysis. Changes in the enzymatic activities of other glycosyltransferases, such as alpha1,3-galactosyltransferase, and alpha-6-D-mannoside beta-1,6 N-acetylglucosaminyltransferase V, did not point to any interaction between ***GnT*** - ***III*** and these enzymes in the transgenic mice, suggesting that this approach may be useful in clinical xenotransplantation.

L14 ANSWER 7 OF 19 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2002:302053 BIOSIS <<LOGINID::20070410>>
DN PREV200200302053
TI ***N*** - ***Acetylglucosaminyltransferase*** - ***III*** .
AU Ikeda, Yoshitaka [Reprint author]; Taniguchi, Naoyuki [Reprint author]
CS Department of Biochemistry, Osaka University Medical School, 2-2 Yamadaoka, Suita, Osaka, 565-0871, Japan
proftani@biochem.med.osaka-u.ac.jp
SO Taniguchi, Naoyuki [Editor]; Honke, Koichi [Editor]; Fukuda, Minoru [Editor]. (***2002***) pp. 80-86. Handbook of glycosyltransferases and related genes. Edition 1. print.
Publisher: Springer-Verlag Tokyo Inc., 3-13 Hongo, Bunkyo-ku, Tokyo, 113-0033, Japan; Springer-Verlag New York Inc., 175 Fifth Avenue, New York, NY, 10010-7858, USA.
ISBN: 4-431-70311-X (cloth).
DT Book
Book; (Book Chapter)
LA English
ED Entered STN: 22 May 2002
Last Updated on STN: 22 May 2002

L14 ANSWER 8 OF 19 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN DUPLICATE 1
AN 2002312012 EMBASE <<LOGINID::20070410>>
TI Truncated, inactive ***N*** - ***acetylglucosaminyltransferase*** ***III*** (GlcNAc-TIII) induces neurological and other traits absent in mice that lack GlcNAc-TIII.
AU Bhattacharyya R.; Bhaumik M.; Raju T.S.; Stanley P.
CS P. Stanley, Dept. of Cell Biology, Albert Einstein College of Medicine, 1300 Morris Park Ave., New York, NY 10461, United States.
stanley@aecom.yu.edu
SO Journal of Biological Chemistry, (19 Jul 2002) Vol. 277, No. 29, pp. 26300-26309. .
Refs: 52
ISSN: 0021-9258 CODEN: JBCHA3
CY United States
DT Journal; Article
FS 008 Neurology and Neurosurgery
021 Developmental Biology and Teratology
022 Human Genetics
029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 19 Sep 2002
Last Updated on STN: 19 Sep 2002
AB ***N*** - ***Acetylglucosaminyltransferase*** ***III*** (GlcNAc-TIII), the product of the Mgat3 gene, transfers the bisecting GlcNAc to the core mannose of complex N-glycans. The addition of this residue is regulated during development and has functional consequences for receptor signaling, cell adhesion, and tumor progression. Mice homozygous for a null mutation at the Mgat3 locus (Mgat3(.DELTA.)) or for a targeted mutation in the Mgat3 gene (previously called Mgat3(neo), but herein renamed Mgat3(T37) because the allele generates inactive GlcNAc-TIII of .apprx.37 kDa) were found to exhibit retarded progression of liver tumors. Matrix-assisted laser desorption/ionization time-of-flight mass spectrometry of neutral N-glycans from kidneys revealed no significant differences, and both mutants showed the expected lack of ***N*** - ***glycan*** species with an additional GlcNAc. However, the two mutants differed in several biological traits. Mgat3(T37/T37) homozygotes in a mixed or 129(SvJ) background were retarded
in growth rate and exhibited an altered leg clasp reflex, an altered gait, and defective nursing behavior. Pups abandoned by Mgat3(T37/T37) mothers were rescued by wild-type foster mothers. None of these Mgat3(T37/T37) traits were exhibited by Mgat3(.DELTA./.DELTA.) mice or by heterozygous mice carrying the Mgat3(T37) mutation. Similarly, no dominant-negative effect was observed in Chinese hamster ovary cells expressing truncated GlcNAc-TIII in the presence of wild-type GlcNAc-TIII. However, compound heterozygotes carrying both the Mgat3(T37) and Mgat3.DELTA. mutations exhibited a marked leg clasp reflex, indicating that in the absence of wild-type GlcNAc-TIII, truncated GlcNAc-TIII causes this phenotype. The Mgat3 gene was expressed in brain at embryonic day 10.5 and thereafter and in neurons of adult cerebellum. The mutant Mgat3 gene was also highly expressed in Mgat3(T37/T37) brain. This may be the basis of the unexpected neurological phenotype induced by truncated, inactive GlcNAc-TIII in the mouse.

L14 ANSWER 9 OF 19 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN DUPLICATE 2
AN 2002416434 EMBASE <<LOGINID::20070410>>
TI Biological consequences of overexpressing or eliminating N-acetylglucosaminyltransferase-TIII in the mouse.
AU Stanley P.
CS P. Stanley, Department of Cell Biology, Albert Einstein College Medicine, Yeshiva University, 1300 Morris Park Avenue, Bronx, NY 10461, United States. stanley@aecom.yu.edu
SO Biochimica et Biophysica Acta - General Subjects, (19 Dec 2002) Vol. 1573, No. 3, pp. 363-368. .
Refs: 39
ISSN: 0304-4165 CODEN: BBGSB3
PUI S 0304-4165(02)00404-X
CY Netherlands
DT Journal; General Review
FS 029 Clinical Biochemistry
LA English

SL English
ED Entered STN: 5 Dec 2002
Last Updated on STN: 5 Dec 2002
AB ***N*** - ***acetylglucosaminyltransferase*** ***III*** (GlcNAc-TIII), a product of the human MGAT3 gene, was discovered as a glycosyltransferase activity in hen oviduct. GlcNAc-TIII transfers GlcNAc in .beta.4-linkage to the core Man of complex or hybrid N-glycans, and thereby alters not only the composition, but also the conformation of the ***N*** - ***glycan*** . The dramatic consequences of the addition of this bisecting GlcNAc residue are reflected in the altered binding of lectins that recognize Gal residues on N-glycans. Changes in GlcNAc-TIII expression correlate with hepatoma and leukemia in rodents and humans, and the bisecting GlcNAc on Asn 297 of human IgG antibodies enhances their effector functions. Overexpression of a cDNA encoding GlcNAc-TIII alters growth control and cell-cell interactions in cultured cells, and in transgenic mice. While mice lacking GlcNAc-TIII are viable and fertile, they exhibit retarded progression of diethylnitrosamine (DEN)-induced liver tumors. Further biological functions of GlcNAc-TIII are expected to be uncovered as mice with a null mutation in the Mgat3 gene are challenged. .COPYRGT. 2002 Elsevier Science B.V. All rights reserved.

L14 ANSWER 10 OF 19 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2003288082 EMBASE <<LOGINID::20070410>>
TI Antibodies that recognize bisected complex N-glycans on cell surface glycoproteins can be made in mice lacking ***N*** - ***acetylglucosaminyltransferase*** ***III*** .
AU Lee J.; Park S.-H.; Stanley P.
CS Dr. P. Stanley, Department of Cell Biology, Albert Einstein College of Medicine, New York, NY 10461, United States. stanley@aecom.yu.edu
SO Glycoconjugate Journal, (1 Mar 2003) Vol. 19, No. 3, pp. 211-219. .
Refs: 38
ISSN: 0282-0080 CODEN: GLJOEW
CY Netherlands
DT Journal; Article
FS 026 Immunology, Serology and Transplantation
029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 31 Jul 2003
Last Updated on STN: 31 Jul 2003
AB The bisecting GlcNAc is transferred to complex or hybrid N-glycans by the action of ***N*** - ***acetylglucosaminyltransferase*** ***III*** (GlcNAc-TIII) encoded by the Mgat3 gene. CHO cells expressing mouse GlcNAc-TIII were shown by matrix-assisted laser desorption ionization (MALDI) mass spectrometry to produce mainly complex N-glycans with the predicted extra (bisecting) GlcNAc. In order to probe biological functions of the bisecting GlcNAc, antibodies that recognize this residue in the context of complex cell surface glycoconjugates were sought. The LEC10 gain-of-function Chinese hamster ovary (CHO) cell mutant that expresses GlcNAc-TIII and complex N-glycans with the bisecting GlcNAc was used to immunize Mgat3(+/+) and Mgat3(-/-) mice. ELISA of whole sera showed that polyclonal antibodies that bound specifically to LEC10 cells were obtained solely from Mgat3(-/-) mice. Fluorescence-activated cell cytometry of different CHO glycosylation mutants and western blotting after glycosidase treatments were used to show that anti-LEC10 cell antisera from Mgat3(-/-) mice recognize cellular glycoproteins with complex N-glycans containing both a bisecting GlcNAc and Gal residues. The polyclonal antibody specificity was similar to that of the lectin E-PHA. IgM-depleted serum containing IgG and IgA antibodies retained full binding activity. Therefore Mgat3(-/-) mice but not wild type mice can be used effectively to produce polyclonal antibodies that specifically recognize glycoproteins bearing complex N-glycans with a bisecting GlcNAc.

L14 ANSWER 11 OF 19 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN DUPLICATE 3
AN 2002046495 EMBASE <<LOGINID::20070410>>
TI A catalytically inactive .beta.1,4- ***N*** - ***acetylglucosaminyltransferase*** ***III*** (***GnT*** - ***III***) behaves as a dominant negative ***GnT*** - ***III*** inhibitor.
AU Ihara H.; Ikeda Y.; Koyota S.; Endo T.; Honke K.; Taniguchi N.
CS N. Taniguchi, Department of Biochemistry, Osaka University Medical School, 2-2 Yamadaoka, Suita, Osaka 565-0871, Japan. proftani@biochem.med.osaka-u.ac.jp
SO European Journal of Biochemistry, (2002) Vol. 269, No. 1, pp. 193-201. .
Refs: 45
ISSN: 0014-2956 CODEN: EJBCAI
CY United Kingdom
DT Journal; Article
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 14 Feb 2002
Last Updated on STN: 14 Feb 2002
AB .beta.1,4- ***N*** - ***Acetylglucosaminyltransferase*** ***III*** (***GnT*** - ***III***) plays a regulatory role in the biosynthesis of N-glycans, and it has been suggested that its product, a bisecting GlcNAc, is involved in a variety of biological events as well as in regulating the biosynthesis of the oligosaccharides. In this study, it was found, on the basis of sequence homology, that ***GnT*** - ***III*** contains a small region that is significantly homologous to both snail .beta.1,4GlcNAc transferase and .beta.1,4Gal transferase-1. Subsequent mutational analysis demonstrated an absolute requirement for two conserved Asp residues (Asp321 and Asp323), which are located in the most homologous region of rat ***GnT*** - ***III*** , for enzymatic activity. The overexpression of Asp323-substituted, catalytically inactive ***GnT*** - ***III*** in Huh6 cells led to the suppression of the activity of endogenous ***GnT*** - ***III*** , but no significant decrease in its expression, and led to a specific inhibition of the formation of bisected sugar chains, as shown by structural analysis of the total N-glycans from the cells. These findings indicate that the mutant serves a dominant negative effect on a specific step in ***N*** - ***glycan*** biosynthesis. This type of 'dominant negative glycosyltransferase', identified has potential value as a powerful tool for defining the precise biological roles of the bisecting GlcNAc structure.

L14 ANSWER 12 OF 19 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN DUPLICATE 4
AN 2001022366 EMBASE <<LOGINID::20070410>>
TI The addition of bisecting N-acetylglucosamine residues to E-cadherin down-regulates the tyrosine phosphorylation of .beta.-catenin.
AU Kitada T.; Miyoshi E.; Noda K.; Higashiyama S.; Ihara H.; Matsuura N.; Hayashi N.; Kawata S.; Matsuzawa Y.; Taniguchi N.
CS N. Taniguchi, Department of Biochemistry, Osaka Univ. Graduate Sch. of Med., 2-2 Yamadaoka, Suita, Osaka 565-0871, Japan. proftani@biochem.med.osaka-u.ac.jp
SO Journal of Biological Chemistry, (5 Jan 2001) Vol. 276, No. 1, pp. 475-480. .
Refs: 35
ISSN: 0021-9258 CODEN: JBCHA3
CY United States
DT Journal; Article
FS 016 Cancer
029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 1 Feb 2001
Last Updated on STN: 1 Feb 2001
AB The enzyme ***GnT*** - ***III*** (.beta.1,4- ***N*** - ***acetylglucosaminyltransferase*** ***III***) catalyzes the addition of a bisecting N-acetylglucosamine (GlcNAc) residue on glycoproteins. Our previous study described that the transfection of ***GnT*** - ***III*** into mouse melanoma cells results in the enhanced expression of E-cadherin, which in turn leads to the suppression of lung metastasis. It has recently been proposed that the phosphorylation of a tyrosine residue of .beta.-catenin is associated with cell migration. The present study reports on the importance of bisecting GlcNAc residues by ***GnT*** - ***III*** on tyrosine phosphorylation of .beta.-catenin using three types of cancer cell lines. An addition of bisecting GlcNAc residues to E-cadherin leads to an alteration in cell morphology and the localization of .beta.-catenin after epidermal growth factor stimulation. These changes are the result of a down-regulation in the tyrosine phosphorylation of .beta.-catenin. In addition, tyrosine phosphorylation of .beta.-catenin by transfection of constitutively active c-src was suppressed in ***GnT*** - ***III*** transfectants as well as in the case of epidermal growth factor stimulation. Treatment with tunicamycin abolished any differences in .beta.-catenin phosphorylation for the mock vis a vis the ***GnT*** - ***III*** transfectants. Thus, the addition of a specific ***N*** - ***glycan*** structure, the bisecting GlcNAc to E-cadherin-.beta.-catenin complex, down-regulates the intracellular signaling pathway, suggesting its implication in cell motility and the suppression of cancer metastasis.

L14 ANSWER 13 OF 19 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN DUPLICATE 5
AN 2001:423635 BIOSIS <<LOGINID::20070410>>
DN PREV200100423635
TI A glycomic approach to the identification and characterization of glycoprotein function in cells transfected with glycosyltransferase genes.
AU Taniguchi, Naoyuki [Reprint author]; Ekuni, Atsuko; Ko, Jeong Heon; Miyoshi, Eiji; Ikeda, Yoshitaka; Ihara, Yoshito; Nishikawa, Atsushi; Honke, Koichi; Takahashi, Motoko
CS Department of Biochemistry, Osaka University Graduate School of Medicine, 2-2 Yamadaoka, Suita, Osaka, 565-0871, Japan proftani@biochem.med.osaka-u.ac.jp
SO Proteomics, (***February, 2001***) Vol. 1, No. 2, pp. 239-247. print. ISSN: 1615-9853.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 5 Sep 2001
Last Updated on STN: 22 Feb 2002
AB The transfection of glycoprotein glycosyltransferase genes into cells leads to modification of both the structure and function of the glycoproteins and as a result, changes in glycome patterns. ***N*** - ***glycan*** branching enzymes hold some promise as a model system for the identification of glycome patterns. Both ***N*** - ***acetylglucosaminyltransferase*** ***III*** and alpha1-6 fucosyltransferase are typical glycosyltransferases, which are involved in the branching of N-glycans. The resulting enzymatic products, bisecting N-GlcNAc and alpha1-6 fucose residues, are no longer modified by other glycosyltransferases and it is a relatively simple task to identify their modification by means of lectins. In this review, the glycome patterns of

glycosyltransferase gene transfectants and the non-transfectants were compared by two-dimensional gel electrophoresis and lectin staining, and the biological significance of the two genes are described. Analyses of glycome patterns by transfecting glycosyltransferase genes will lead to new fields of study in the area of postgenome research.

L14 ANSWER 14 OF 19 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN DUPLICATE 6
AN 2000:305222 BIOSIS <<LOGINID::20070410>>
DN PREV200000305222
TI Comparative study of the N-glycans of human monoclonal immunoglobulins M produced by hybridoma and parental cells.
AU Fukuta, Kazuhiro [Reprint author]; Abe, Reiko; Yokomatsu, Tomoko; Kono, Naoko; Nagatomi, Yuji; Asanagi, Mineko; Shimazaki, Yukio; Makino, Tadashi
CS Life Science Laboratory, Mitsui Chemicals, Inc., 1144, Togo, Mobara, Chiba, 297-0017, Japan
SO Archives of Biochemistry and Biophysics, (***June 1, 2000***) Vol. 378, No. 1, pp. 142-150. print.
CODEN: ABBIA4. ISSN: 0003-9861.
DT Article
LA English
ED Entered STN: 19 Jul 2000
Last Updated on STN: 7 Jan 2002
AB Cell-cell hybridization is one method of establishing cell lines capable of producing an abundance of antibodies. In order to clearly characterize antibodies produced by hybridomas, the influence of cell-cell hybridization on the glycosylation of produced antibodies should be studied. In this report, we describe structural changes of the N-glycans in immunoglobulin M (IgM) produced by a hybridoma cell line termed 3-4, which was established through hybridization of an IgM-producing Epstein-Barr virus transformed human B-cell line termed No. 12, and a human myeloma cell line termed P109. We analyzed the structures of sugar chains on the constant region of the mu-chain of IgMs produced by parental No. 12 cells and hybridoma 3-4 cells. In both parental cells and hybridoma cells, the predominant structures at Asn171, Asn332, and N395 were fully galactosylated biantennary complex types, with or without core fucose and/or bisecting GlcNAc. However, the amount of bisecting GlcNAc was markedly decreased in the hybridoma cells. Therefore, the activity of UDP-N-acetylglucosamine:beta-D-mannoside beta-1,4-N-acetylglucosaminyltransferase (***GnT*** - ***III***) responsible for the formation of bisecting GlcNAc was measured in parental cells and hybridoma cells. No. 12 cells showed some ***GnT*** - ***III*** activity, whereas P109 cells showed no such activity. The corresponding level of activity observed in hybridoma 3-4 cells was much lower than that in No. 12 cells. The above results demonstrated a reduction in the intracellular activity of ***GnT*** - ***III*** in the hybridoma cells, which was largely due to the influence of P109 cells. Moreover, the sugar chain structures of IgMs produced by the cells reflected the level of ***GnT*** - ***III*** activity.

L14 ANSWER 15 OF 19 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN DUPLICATE 7
AN 97120373 EMBASE <<LOGINID::20070410>>
DN 1997120373
TI Overexpression of ***N*** - ***acetylglucosaminyltransferase*** ***III*** disrupts the tyrosine phosphorylation of Trk with resultant signaling dysfunction in PC12 cells treated with nerve growth factor.
AU Ihara Y.; Sakamoto Y.; Mihara M.; Shimizu K.; Taniguchi N.
CS N. Taniguchi, Dept. of Biochemistry, Osaka University Medical School, 2-2 Yamadaoka, Suita, Osaka 565, Japan
SO Journal of Biological Chemistry, (1997) Vol. 272, No. 15, pp. 9629-9634. .
Refs: 42
ISSN: 0021-9258 CODEN: JBCHA3
CY United States
DT Journal; Article
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 20 May 1997
Last Updated on STN: 20 May 1997
AB .beta.-1,4- ***N*** - ***Acetylglucosaminyltransferase*** ***III*** (***GnT*** - ***III*** : EC 2.4.1.144) is a pivotal glycosyltransferase which participates in branch formation by catalysis of the synthesis of a bisecting GlcNAc structure in N-glycans. These structures are thought to be one of the unique features of the N- glycans of neural tissues. To examine the intracellular role of ***GnT*** - ***III*** expression and its product in neural cells, its gene was overexpressed in rat pheochromocytoma PC12 cells which normally express a low level of ***GnT*** - ***III*** . In the ***GnT*** - ***III*** gene-transfected cells, lectin blot analysis showed that some glycoproteins showed increased levels of bisecting GlcNAc structures. Following treatment with nerve growth factor (NGF) the control cells showed neurite outgrowth for differentiation whereas the transfectants showed no morphological response or change in the rate of cell growth. Transient tyrosine phosphorylation of the Trk/NGF receptor was detected at 5-15 min after NGF treatment in control cells, but not detected in the ***GnT*** - ***III*** gene- transfected cells despite the intact binding of NGF to the cells. Moreover the dimerization of Trk with NGF treatment was not induced in the ***GnT*** - ***III*** transfectant as compared with the dimerization seen in control cells. These results indicate that overexpression of ***GnT*** - ***III*** gene in PC12 cells affects some functions of glycoprotein receptors such as Trk by alteration of ***N*** - ***glycan*** structures, and results in changes in the intracellular signaling pathway of tyrosine phosphorylation modified by NGF.

L14 ANSWER 16 OF 19 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1997:264742 CAPLUS <<LOGINID::20070410>>
DN 126:275253
TI Remodeling of ***N*** - ***glycan*** structures by ***GnT*** - ***III*** gene and its biological consequences
AU Ihara, Yoshito; Taniguchi, Naoyuki
CS Med. Sch., Osaka Univ., Suita, 565, Japan
SO Igaku no Ayumi (***1997***), 180(10), 649-652
CODEN: IGAYAY; ISSN: 0039-2359
PB Ishiyaku
DT Journal; General Review
LA Japanese
AB A review with 8 refs., on N-acetyl-glucosaminyltransferase III (***GnT*** - ***III***)-mediated glycoprotein sugar chain remodeling and biol. function, and role of ***GnT*** - ***III*** -mediated glycoprotein sugar chain remodeling in pathogenesis of liver diseases.

L14 ANSWER 17 OF 19 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN DUPLICATE 8
AN 97064228 EMBASE <<LOGINID::20070410>>
DN 1997064228
TI Isolation, characterization and inactivation of the mouse Mgat3 gene: The bisecting N-acetylglucosamine in asparagine-linked oligosaccharides appears dispensable for viability and reproduction.
AU Priatel J.J.; Sarkar M.; Schachter H.; Marth J.D.
CS J.D. Marth, Howard Hughes Medical Institute, University of California, 9500 Gilman Drive, San Diego, La Jolla, CA 92093, United States
SO Glycobiology, (1997) Vol. 7, No. 1, pp. 45-56. .
Refs: 51
ISSN: 0959-6658 CODEN: GLYCE3
CY United Kingdom
DT Journal; Article
FS 002 Physiology
021 Developmental Biology and Teratology
029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 18 Mar 1997
Last Updated on STN: 18 Mar 1997
AB The biosynthesis of complex asparagine (N)-linked oligosaccharides in vertebrates proceeds with the linkage of N-acetylglucosamine (GlcNAc) to the core mannose residues. UDP-N-acetylglucosamine: .beta.-D-mannoside .beta.1-4 ***N*** - ***acetylglucosaminyltransferase*** ***III*** (GlcNAc-TIII, EC2.4.1.144) catalyzes the addition of GlcNAc to the mannose that is itself .beta.1-4 linked to underlying N-acetylglucosamine. GlcNAc-TIII thereby produces what is known as a 'bisecting' GlcNAc linkage which is found on various hybrid and complex N-glycans. GlcNAc-TIII can also play a regulatory role in ***N*** - ***glycan*** biosynthesis as addition of the bisecting GlcNAc eliminates the potential for .alpha.-mannosidase-II, GlcNAc-TIII, GlcNAc-TIV, GlcNAc-TV, and core .alpha.1-6-fucosyltransferase to act subsequently. To investigate the physiologic relevance of GlcNAc-TIII function and bisected N-glycans, the mouse gene encoding GlcNAc-TIII (Mgat3) was cloned, characterized, and inactivated using Cre/loxP site-directed recombination. The Mgat3 gene is highly conserved in comparison to the rat and human homologs and is normally expressed at high levels in mammalian brain and kidney tissues. Using fluorescence in situ hybridization (FISH), the Mgat3 gene was regionally mapped to chromosome 15E11, near the Scn8a sodium channel gene at 15F1. Following homologous recombination in embryonic stem cells and Cre mediated gene deletion, Mgat3-deficient mice were produced that lacked GlcNAc-TIII activity and were deficient in E4-PHA visualized GlcNAc-bisected N-linked oligosaccharides. Nevertheless, GlcNAc-TIII deficient mice were found to be viable and reproduced normally. Moreover, such mice exhibited normal cellularity and morphology among organs including brain and kidney, No alterations were apparent in circulating leukocytes, erythrocytes or in serum metabolite levels that reflect kidney function. We thus find that GlcNAc-TIII and the bisecting GlcNAc in N-glycans appear dispensable for normal development, homeostasis and reproduction in the mouse.

L14 ANSWER 18 OF 19 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN DUPLICATE 9
AN 96008963 EMBASE <<LOGINID::20070410>>
DN 1996008963
TI Effects of dibutyryl cAMP and bromodeoxyuridine on expression of N-acetylglucosaminyltransferases III and V in GOTO neuroblastoma cells.
AU Ihara Y.; Nishikawa A.; Taniguchi N.
CS Department of Biochemistry, Osaka University Medical School, 2-2 Yamadaoka,Suita, Osaka 565, Japan
SO Glycoconjugate Journal, (1995) Vol. 12, No. 6, pp. 787-794. .
ISSN: 0282-0080 CODEN: GLJOEW
CY United Kingdom
DT Journal; Article
FS 008 Neurology and Neurosurgery
016 Cancer
029 Clinical Biochemistry
052 Toxicology
LA English

SL English
ED Entered STN: 6 Feb 1996
Last Updated on STN: 6 Feb 1996
AB The sugar chain structures of the cell surface change dramatically during cellular differentiation. A human neuroblastoma cell line, GOTO, is known to differentiate into neuronal cells and Schwannian cell-like cells on treatments with dibutyryl cAMP and bromodeoxyuridine, respectively. We have examined the expression of UDP-N-acetylglucosamine: .beta.-D-mannoside .beta.-1,4N-acetylglucosaminyltransferase III (***GnT*** - ***III*** : EC 2.4.1.144) and UDP-N-acetylglucosamine: .alpha.-6-D-mannoside .beta.-1,6N-acetylglucosaminyltransferase V (GnT-V: EC 2.4.1.155), two major branch forming enzymes in ***N*** - ***glycan*** synthesis, in GOTO cells on two distinct directions of differentiation. In neuronal cell differentiation, ***GnT*** - ***III*** activity showed a slight increase during initial treatment with Bt2cAMP for 4 days and decreased drastically after the fourth day, but the mRNA level of ***GnT*** - ***III*** did not show a decrease but in fact a slight increase. GnT-V activity increased to approximately two- to three-fold the initial level with increasing mRNA level after 8 days, and lectin blot analysis showed an increase in reactivity to Datsura stramonium (DSA) of the immunoprecipitated neural cell adhesion molecule (NCAM). In Schwannian cell differentiation, the activity and mRNA level of ***GnT*** - ***III*** showed no significant change on treatment with BrdU. GnT-V activity also showed no change in spite of the gradual increase in the mRNA level. These results suggest that the activation of GnT-V during neuronal cell differentiation of GOTO cells might be a specific change for branch formation in N-glycans, and this affects the sugar chain structures of some glycoproteins such as NCAM.

L14 ANSWER 19 OF 19 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 1995:527802 BIOSIS <<LOGINID::20070410>>
DN PREV199598542102
TI Synthesis of pentasaccharide analogues of the ***N*** - ***glycan*** substrates of ***N*** - ***acetylglucosaminyltransferase*** ***III*** , IV and V using tetrasaccharide precursors and recombinant beta-(1 fwdarw 2)-N-acetylglucosaminyltransferase II.
AU Reck, Folkert; Meinjohanns, Ernst; Tan, Jenny; Grey, Arthur A.; Paulsen, Hans; Schachter, Harry [Reprint author]
CS Res. Inst., Hosp. Sick Children, Toronto, ON M5G 1X8, Canada
SO Carbohydrate Research, (***1995***) Vol. 275, No. 2, pp. 221-229. CODEN: CRBRAT. ISSN: 0008-6215.
DT Article
LA English
ED Entered STN: 14 Dec 1995
Last Updated on STN: 27 Jan 1996
AB Recombinant human UDP-GlcNAc:alpha-Man-(1 fwdarw 6)R beta-(1 fwdarw 2)-N-acetylglucosaminyltransferase II (EC 2.4.1.143, GlcNAc-T II) was produced in the Sf9 insect cell/baculovirus expression system as a fusion protein with a (His)-6 tag and partially purified by affinity chromatography on a metal chelating column. The partially purified enzyme was used to catalyze the transfer of GlcNAc from UDP-GlcNAc to R-alpha-Man(1 fwdarw 6)(beta-GlcNAc(1 fwdarw 2)alpha-Man(1 fwdarw 3))beta-Man-O-octyl to form beta-GlcNAc(1 fwdarw 2)R-alpha-Man(1 fwdarw 6)(beta-GlcNAc(1 fwdarw 2)alpha-Man(1 fwdarw 3))beta-Man-O-octyl where there is either no modification of the alpha-Man(1 fwdarw 6) residue (7), or where R is 3-deoxy (8), 4-deoxy (9) or 6-deoxy (10). The yields ranged from 64-80%. Products were characterized by 1H and 13C nuclear magnetic resonance spectroscopy and fast atom bombardment mass spectrometry. Compounds 7-10 are pentasaccharide analogues of the biantennary ***N*** - ***glycan*** substrates of N-acetylglucosaminyltransferases III, IV and V.

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